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XVII.

A SURGICAL CONSIDERATION OF THE UPPER PARANASAL CELLS.*

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I propose a surgical procedure for the upper paranasal cells which cuts downward and forward. All other procedures, so far as I know, cut more or less upward and backward.

Anatomically the paranasal cells are designated according to which meatus of the nose they enter. This is more or less an anterior and posterior subdivision, and for the purposes of diagnosis this must remain unchanged. For surgical purposes, however, it seems to me that they may advantageously be thought of as upper and lower, the latter being the antrum of Highmore, the former being the remaining cells. This subdivision establishes a horizontal dividing line on the lateral wall at about its middle. Successful surgery of the antrum must leave a drain at the level of the nasal floor. The lower-

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most part of the upper cells is along the line separating them from the antrum—i. e., the horizontal midline. Successful surgery of the upper sinuses, however, is not achieved by a drain along this line, for the reason that there are usually cells which are limited to a part far above the midline. Successful surgery of the upper sinuses must lay open for drain the upper cells in their entirety, beginning at or very close to the cribriform plate of the ethmoid—the roof of the nose—opening at the same time the lowermost of these cells—that is, removing the lateral wall of the nose with the middle and upper turbinates above the horizontal midline.

This procedure, it will be readily conceived, may be conservative or may be made radical. The former consists of the removal of the turbinate and the very innermost layer of the capsule of the ethmoid. The radical operation consists in removing the capsule "part and parcel," including the orbital plate, when desirable for drainage of the orbit into the nose, at any or all its points, from its anterior limit to its apex.

To this end particularly was directed the understanding mind of H. P. Mosher when he proposed the "surgery of the ethmoidal labyrinth." This procedure must of necessity be a radical one, although he does not recommend it for the extreme of radical—opening the orbit.

Also to this end was directed the fearless effort of W. L. Ballenger when he proposed the exenteration of the ethmoid.

It seems to me that the procedure I herewith submit has some advantages that neither of the others offer. First, Mosher's operation cannot (as I understand it) be made conservative. By this I mean the simple freeing of the outlets of the cells, leaving their walls otherwise intact. And the more completely it is executed the more risk it must needs involve. In the hands of anyone less familiar with it than Dr. Mosher, it must become more serious the closer he approaches the uppermost nasal limits.

Second, Ballenger's ethmoidal exenteration requires as its primary requisite, soft bone—that is, bone that has been softened by disease—and, furthermore, it requires a lateral wall

that is not a plane. Should the lower half of the lateral wall extend outward in a way more or less under the ethmoidal capsule, the special knife he so ingeniously devised may be readily engaged from behind and cut more or less into the capsule in its forward and upward pull. I cannot, however, conceive of it opening ethmoidal cells anterior to the uncinate process, because the direction of the blade changes in this part of the nose so as to defeat this; and the uncinate process is usually, if not always, so hard that it will cast the knife back into the free space of the nasal fossa. It will turn it off as a chisel may be turned off by a nail in a piece of wood. Should the lateral wall be approximately a plane with the turbinate set on it, the knife will remove the posterior four-fifths of the turbinate only, unless the wall be exceedingly soft, when it might bite into the capsule. But for a nostril with many polyps and swollen capsule made of cells filled with pus, Ballenger's exenteration has many advantages—e. g., the instrument need be introduced into the nose only once, which helps much with terrified patients; and it may be done quickly. For these reasons I prize it and use it especially where the case is ethmoidal without frontal or sphenoidal elements.

The procedure I describe has been satisfactory in my hands for thirteen years. It may be limited for the frontal sinus, providing a very high cut of the middle turbinate, which I have frequently mentioned in various texts as a "cribriform turbinectomy." The cut is actually two or two and one-half millimeters from the cribriform plate, and may be extended to the most anterior limit of the infundibulum. Or this very high cut may be carried backward to include the capsule of the ethmoid, under which conditions not only is the middle turbinate removed, but the uppermost line, and usually all the other ethmoidal cells are opened wide into the nasal cavity. And when desirable the entire anterior wall of the body of the sphenoid, from its uppermost limits, with all its postethmoidal association and much of its floor, may be removed. In my judgment and experience it is the technic that most often may be trusted to open all of the cells, regardless of unusual or

anomalous positions. I know, however, that cells may exist that not only cannot be opened from the nose, but cannot be located by any means at present at our command. They can only be recognized in the cadaver, and then only by complete dissection. Everyone who has observed a reasonable number of specimens must have seen such cells and have been struck by the futility that would attend any surgical effort to reach them; and everyone of reasonably wide experience has met cases where all efforts had failed to find and treat such cells. But there are many other cells that are placed in positions more or less unusual that will be opened by a technic which has for its primary plan an incision which will skirt the cribriform plate and remove the middle turbinate at its most anterior as well as its uppermost limits, and extend into the sphenoid body at its uppermost part, regardless of the natural opening, and then be extended downward until it has cut through its floor or found it to be impenetrable. I have here avoided the designation of sphenoid sinus, because such a term does not always comprehend the full body of the sphenoid. It (the body) may be subdivided and shared by a post-ethmoidal cell. And it is this variation which I believe to be the most pernicious from the clinical side (for headache and optic nerve lesions) as well as anatomically far more frequent than is at present recognized clinically. This variation will (it seems to me) be satisfactorily dealt with by this proposed technic—practically every time it is met. Also prefrontal ethmoidal cells will be opened into the nose as a rule, if not uniformly.

The Operation.—In 1907 I published an elementary text in which I described a surgical method which was at that time novel. It consisted in approaching the turbinate from above on its inner side. Prior to that all surgical approach was from below laterally upward, removing it by scissors or snare or a combination of these or such working instruments. I described a knife consisting of a handle, a shaft and a cutting end turned at a right angle to the shaft and sharpened so as to cut on the inside of the right angle, i. e., on the pull. It was

also sharpened on a face parallel to the shaft, and at the same time gave it far more strength than a hook must have were it sharpened on its concavity. I selected this shape in preference to a hook for these reasons, and the fact that this rectangular form is much stronger than a hook; and that great strength is necessary for the tasks to which this knife is put. The knife, although possessing great strength, is so small that it may readily enter spaces which larger instruments could not. I believe it to be the smallest nasal instrument possessing great strength.

Surgical Procedure.—A glance at the lateral wall shows that the higher and the more forward the infundibulum is opened, the wider and freer will be the inlet to the frontal sinus usually. At the same time prefrontal ethmoidal cells will usually be opened by such an incision. For these reasons it has been advantageous in my hands to introduce the knife sagittally under the anterior third of the middle turbinate as far back as the uncinate process, as high as the cribriform plate, with its cutting edges facing forward. It is then rotated somewhat inward and drawn forward and downward. (Fig 1.) Considerable forward pressure is kept on it while it is being drawn downward. In this way the foremost and uppermost nasal wall of the capsule of the ethmoid (with the attachment of the middle turbinate) is cut open and the prefrontal cells of the ethmoid are thereby thrown open, as the nasal wall of these cells is usually constituted by this part of capsule with the attachment of the turbinate. This must be considered the conserative technic. By these means, however, is put an outlet to the usual frontal sinus that comprehends the dimensions of the usual infundibulum—i. e., one centimeter anteroposteriorly and one-half centimeter laterally. Should this size be reduced to such a degree as to require some procedure more radical, the knife is then engaged in the hiatus semilunaris, with its cutting edges facing forward or downward—usually in the uppermost limit. By a down pull the uncinate process will be removed and the knife may finally be engaged in the floor of the anterior part of the frontal

sinus. (Fig. 12.) This is usually so thick and hard that it is with difficulty or impossibility handled by this knife. A large enough opening, however, is usually secured to admit a Good rasp or a burr. The burr is then introduced into the sinus and put in motion with a forward and downward pull. In this way I have slightly modified Ingals' ideal frontal sinus technic. This stroke of the right angle knife it will be seen is safe, as it cuts away from danger; or the knife in position, cutting edges facing upward and forward, is likewise safe with a downward pull, as it also cuts away from danger. It will cut into the buttress of the lower anterior part of the floor of the sinus.

For the ethmoidal district the knife is reintroduced along the cribriform plate—its smooth elbow with a gentle sense of touch, recognizing the smooth uppermost limit of the nose—to a distance of one centimeter. In this position its two cutting edges face downward. It is now rotated till its cutting edges point outward and slightly upward. The upward turn is added to the outward for the reason that the outer (horizontal)position permits of a straight line pull downward and forward, and this direction is not easily changed. This is at once away from the cribriform plate. The slight upward turn keeps the line of incision much more nearly the horizontal, despite the fact that the pull must needs be downward and forward or forward and downward. The safety of this cut is provided by two factors—i. e., the cells of the ethmoid in their extensions into the roof of the orbit rise at once above the cribriform plate; and the length of the cutting arm of the knife, which should be two millimeters, is too short to cut through thickness of the nasal orbital thickness of the ethmoid capsule under any circumstances. This incision is the first of two or three similar ones. (Figs. 2 and 3.) I prefer to divide it into two or three because I can keep my bearing upon the cribriform plate more satisfactory by these shorter cuts than in one longer cut.

Now the operative procedure is to be directed by the judgment of the surgeon. In my judgment the only turbinectomy, according to my experience, should be a cribriform or infundi-

bular turbinectomy—i. e., one that opens the infundibulum to its widest possibilities, for the reason that a lower cut leaves a stump that is physiologically worthless and surgically difficult of later manipulation. This is not in reality a stump, although in former years I considered it such. It is that part of the ethmoidal capsule which makes the inner portion of the final entrance of the frontal sinus into the nose—the anterior part being the uncinate process, the posterior part being the bulla ethmoidalis, the external and internal parts being ethmoid capsule. The removal of this inner part as high as possible usually gives an inlet to the frontal sinus six millimeters anteroposteriorly by three millimeters laterally. Some frontals enter the nose by a devious* channel and cannot be considered as usual. If this upper internal part of the frontal inlet be not removed at the primary operation, it seems to me to be unfortunate, as a secondary operation will almost surely wound the epithelial surfaces of the frontal inlet and then be followed by connective tissue development which will block the inlet. The primary high cut is usually accomplished without that result, leaving the hiatus semilunaris and infundibulum free of scar tissue. The secondary operation is usually of necessity done with more trauma to the hiatus, regardless of whatever technic be employed. For this reason I emphasize the advisability of doing the foremost cribriform cut primarily if the anterior half of the turbinate is touched at all surgically, regardless of whatever else is to be done surgically.

The cutting edges of the knife are now turned downward, or downward and slightly outward (Fig. 4), and the incision is placed superficial (internally), or may be placed as deep (externally) in the capsule of the ethmoid as the judgment of the surgeon directs. For the simplest subacute suppuration cases the superficial incision suffices. For most radical—e. g., an orbital phlegmon back of the transverse meridian of the eye—the incision goes through the capsule into the orbit in its posterior half. This is readily and quickly done. The detached mass is then by means of the same knife, which may

*Hyman and Ritter.

be turned somewhat transverse, pushed downward into the nose, where a snare may be put around it and cut it from its attachments. (Fig. 5.) The procedure may be varied by a superficial removal of the turbinate and then the excision of the capsule to any extent desired. In my practice I have had twenty orbital phlegmons, produced by nasal sinus ruptures, twelve of which I opened into the nose in this way back of the transverse meridian. In my experience this has far surpassed the results of external orbital drainage, inasmuch as it drains by the same route that infected the orbit.

Should the sphenoidal and postethmoidal cells also need the surgeon's care, I then (after the snare amputation of the middle turbinate) pass the angular knife very gently backward along the cribriform plate, with its cutting edges downward. In this way its smooth round elbow glides along the cribriform plate until it meets the anterior face of the body of the sphenoid. This it will be seen is the uppermost point of the face, and is independent of the natural opening of the sphenoid sinus, which is usually well below this point.* Gentle pressure is now made on the face of the sphenoid and the knife inclined slightly toward the septum. The first stroke perforates the usual sphenoidal face and cuts somewhat downward. The knife, cutting edges downward, is then introduced very gently into the opening so made and very gently extended as far back as the cell permits. It is rotated fifteen degrees inward, and downward with any degree of force that may be needed. This incision will extend from the junction of the cribriform plate with the body of the sphenoid downward a varying distance along the septum nasi, depending on the thickness and hardness of the bone. Should this incision not have reached the floor of the sphenoid, another similar one usually does. (Fig. 6.) The knife, edges down, is again passed along the cribriform plate and very carefully introduced back into the uppermost limit of the opening already made. It is then rotated thirty degrees outward and brought out forward and downward. (Fig. 7.) This incision includes a considerable part of the wall separating the postethmoidal from the sphenoidal cell, and it has the advantage of beginning at the uppermost limit of the sphenoidal face. It has a distinct advantage

*Loeb.

of all methods which begin their procedure in or from the natural opening of the sphenoid, for the reason that often a postethmoidal cell occupies a part of the body of the sphenoid and is placed above the sphenoidal cell. Fig. 13 shows such a cell which would be opened by this technic and would probably not be opened by any other technic now known. Such cells are exceedingly hard to deal with surgically and sometimes equally hard or impossible to find. Fig. 14B shows such a cell placed all above the line of the cribriform plate. This cell would not be opened by this technic, and I do not believe it could be opened by any other technic. Moreover, I do not believe it could be recognized in life by the present means of investigation. Some such cells, however, have a lateral and downward extension which is opened by the second stroke of the knife, which is again introduced along the cribriform plate and inserted into cavity in the body of the sphenoid, turned thirty degrees outward and drawn forward and downward.

This second cut usually takes the postethmoidal nasal wall, as well as most of the lateral part of the sphenoidal face, and often the mass may be removed in one piece. For this purpose I use the Knight forceps (Figs. 9 and 10), made, however, for this particular purpose, being two centimeters longer than the original model. These forceps are then introduced into the cavity of the sphenoid (Fig. 8), and so the floor of the sphenoid is removed in part. Everyone knows, however, that the floor often is so thick and hard that it cannot be removed by forceps. A drill which will do such service may be advantageous.

In the performance of the postethmoidal sphenoidal portion of the technic I prefer a knife smaller and slightly different from that used in the anterior portion, for the reasons that it is thereby rendered much stronger; it cannot be broken in any of the service it may be put into, and secondly, a knife 1.5 millimeters inside cutting edge cannot inadvertently cut into the orbit in this service unless wielded by some one neglecting the proper performance of the operation and totally ignorant of the anatomy here; and third, I feel that the angle of the sphenoid knife should not have turned the full ninety degrees from shaft, because such a knife could be only with

difficulty engaged in the face of the sphenoid; an angle sixty-eight degrees off the shaft, which will make the cutting edge on the inside of the angle one hundred and twelve degrees, will bite readily into the face; and fourth, a small knife 2.5 millimeters in its entire cross measurement permits a much closer approach to the tip of the sphenoid face. One 6 millimeters could not get so close by 3.5 millimeters. In this service a hook sharpened on its concavity would be much less advantageous, because, if bent to a right angle, it would not bite into the face, and if in sixty-eight to one hundred and twelve degrees shape it would not execute the work so well and its point would break easily. It would then take the shape of the knife now proposed.

I feel that special emphasis should be laid upon the necessity of the surgeon knowing what are the correct models of these knives. For this reason I submit accurate drawings. Too large a knife has the disadvantage (1) of taking up space unnecessarily and of (2) biting more bone than it can cut, and (3) of possibly cutting inadvertently into the orbit, and (4) of being much more apt to have its angle cutting portion broke off in the operation. In this connection it must be emphasized that it is absolutely necessary that the knife be not tempered too hard. Should it be too hard it will always break with the work put on it in this operation. The instrument maker must specially understand this. It is far better to have it too soft, and though its cutting edges may not be as lasting, it will not break.

SUMMARY.

The intranasal surgery of the upper cells may be performed by this method in any part or the whole as conservatively or as radically as desired. The ability to place the incision two millimeters below the cribriform plate with safety in any part or in the whole length of its extent seems to me to be most advantageous, and not a small part of this is the power to extend this incision to the foremost limit of the infundibulum, thereby opening the inlet of the frontal to its widest natural possibilities. It is most desirable to preserve the natural inlet here, and this is done by a cribriform turbinectomy which leaves undisturbed the histologic epithelial covering of the normal inlet—i. e., the uncinate process, the bulla ethmoidalis,

the hiatus semilunaris and infundibulum—regardless of the anatomic variations of the frontal inlet. Should these parts be wounded, as in a curettement, the resultant scar tissue blocks the inlet. The angle knife removes by cutting any desired tissue with the least possible trauma to the surroundings. In the sphenoidal district it opens uppermost and lowermost possible parts of the wall, which has the advantage sometimes of opening also a postethmoidal cell which may occupy part of the body of the sphenoid. (Such a cell is often the cause of the entire clinical picture). The angle knife is so small that it takes up the minimum of room, and so leaves the small field open to the best vision possible. Its execution is always in the direction away from the danger zone. I have so far not seen such satisfactory postethmoidal surgery by other methods. (This district seems to me the most dangerous of all, for on the outer upper aspect runs the optic nerve, and above is the optic chiasm and cranial cavity). Satisfactory execution here is necessary, particularly in eye lesions.

The entire performance may be accomplished within a short time. I have often finished the high frontal ethmoidal and sphenoidal combined operation in two minutes (including the postorbital opening on one occasion). A septum nasi deflected into the affected side may add troubles for the surgeon. I have in such cases where the middle turbinate was not visible from in front in any part used a bivalve speculum specially constructed for the purpose. Just as Killian elongated the blades of the primary bivalve for his needs in septum resection, I have elongated them still more and widened them for my needs here (Fig. 7). The speculum should be made of tempered steel blades, knife-like, thin, with handles long enough to give leverage to dislocate the entire septum into the opposite nostril. It should have a set screw, for the pressure required is much and the hand gets tired holding it. It should have blades nine millimeters wide, for narrower ones, such as are supplied with Killian's, often do not give sufficient view. They should be eighty-eight centimeters long, for shorter ones (Killian's) sometimes fail to reach the posterior field of operation.

The knives are also made right and left (Figure 16). Some of my associates prefer these. My own preference is for the

single, straight, original model. I recommend an extra two and one-half centimeters long shaft for use with the long blade sphenoidal speculum. A strong, large handle ought always to be supplied for the knife.

A right-hand surgeon makes his turbinate cuts for the left side, cutting edges in the place of the handle. For the right side the cutting edges are turned one-quarter (ninety degrees) into right nostril. This makes them horizontal or transverse when the handle is on the middle line.

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FIGURE 1.

On the right the angle knife is shown in the sagittal plane, ready for insertion under the middle turbinate. On the left it is shown in position for the forward and downward cut. It has been introduced into the infundibulum and rotated thirty degrees inwards— i. e., toward the septum nasi. The heavy line a-b shows diagrammatically the line of this first cut, which may be accomplished in one stroke or two, according to the surgeon's election. (I prefer two strokes usually.)

FIGURE 2.

Shows the angle knife introduced between the septum and middle turbinate. It has passed one centimeter back of the first cut along the cribriform plate. The dotted line X shows it to have been rotated outward until its cutting edges point thirty degrees above the horizontal. The tip of the nose has, at the asme time, been forcibly elevated. The heavy dotted line a-b shows the line of cut.

FIGURE 3.

Is the same as Figure 2, with the difference that the knife has been passed the second time along the cribriform plate to a distance one centimeter back of the first cut.

FIGURE 4.

Shows the knife introduced sagittally under (lateral to) the middle turbinate for the purpose of cutting into the capsule of the ethmoid. The dotted lines 1-2-3 show the successive cuts from above downward. These cuts may be readily made to enter the orbit by inclining the cutting edges thirty degrees outward. In the conservative employment of this technic, this employment of the knife may be omitted.

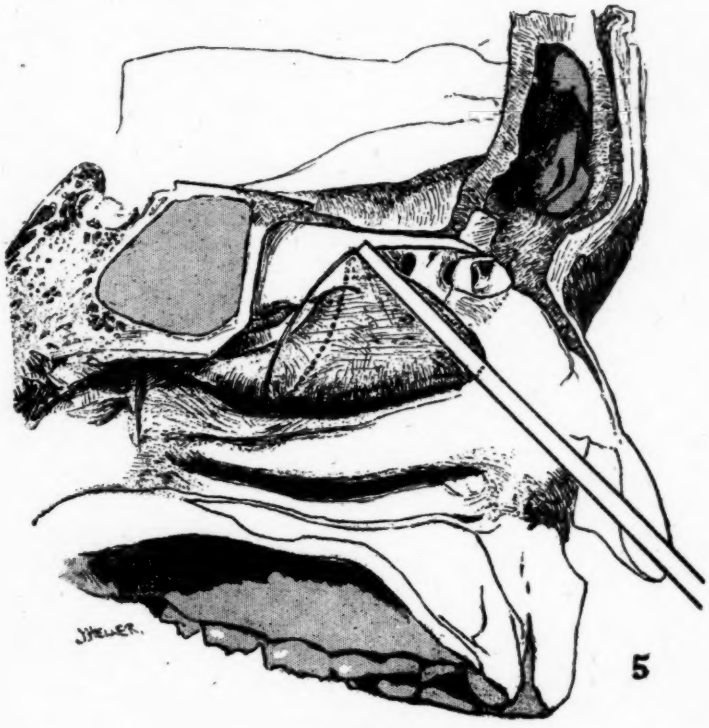

FIGURE 5.

Shows the snare loop placed around part detached.

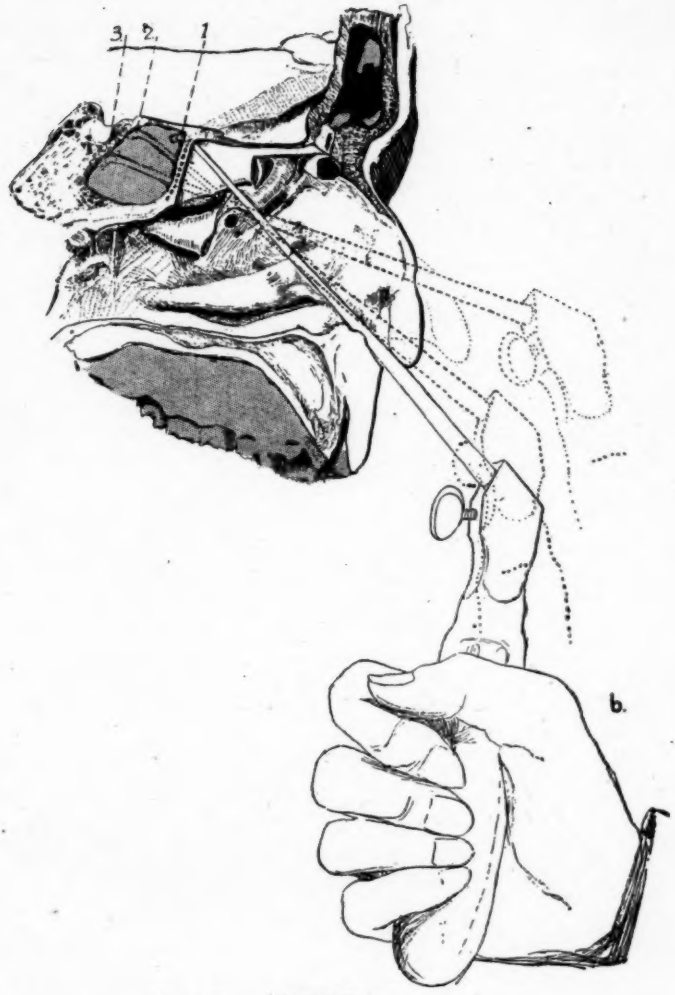

FIGURE 6.

Shows knife 1 passed backward along cribriform plate, penetrating anterior face of sphenoid at its top. The dotted lines 2 and 3 show knife passed back to posterior wall of cell and to have been rotated twenty degrees inward, cutting downward to hug the septum nasi.

FIGURE 7.

Shows knife to have been reintroduced through uppermost part of sphenoidal cut, as shown in Figure 6, to posterior wall of cell. It is then rotated thirty degrees outward (X) and carefully drawn forward and (necessarily) downward. The introduction of the knife to the posterior wall of the cell of its uppermost part insures the opening of many of the postethmoid cells that are developed into the sphenoid body. I believe this to be very important and one of the advantages to be secured by this technic.

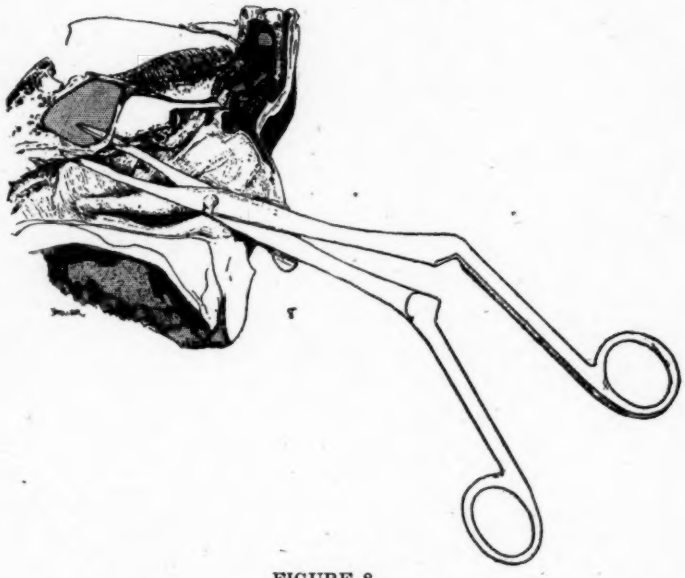

FIGURE 8.

Shows the Knight forceps (made especially for this purpose three centimeters longer than the original model, because this added length is often required) introduced into the opening in the anterior face of the sphenoid body ready to bite out the anterior half of the floor of the body. This is oftentimes easily done, but at other times the floor is too thick to be removed this way. It is removed by a rotatory movement.

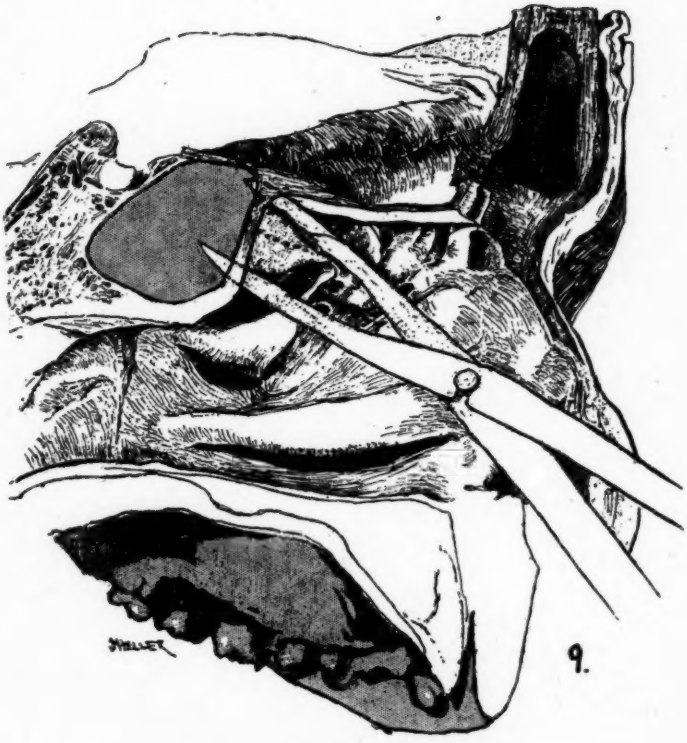

FIGURE 9.

Shows the Knight forceps in position to bite out the postethmoidal wall which has been cut loose, as shown in Figure 7.

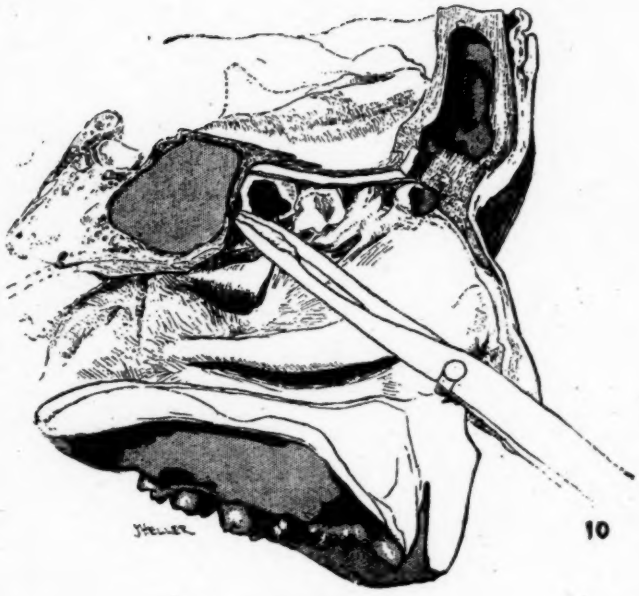

FIGURE 10.

Shows postethmoidal wall in grasp of forceps. It is finally detached by a rotatory movement

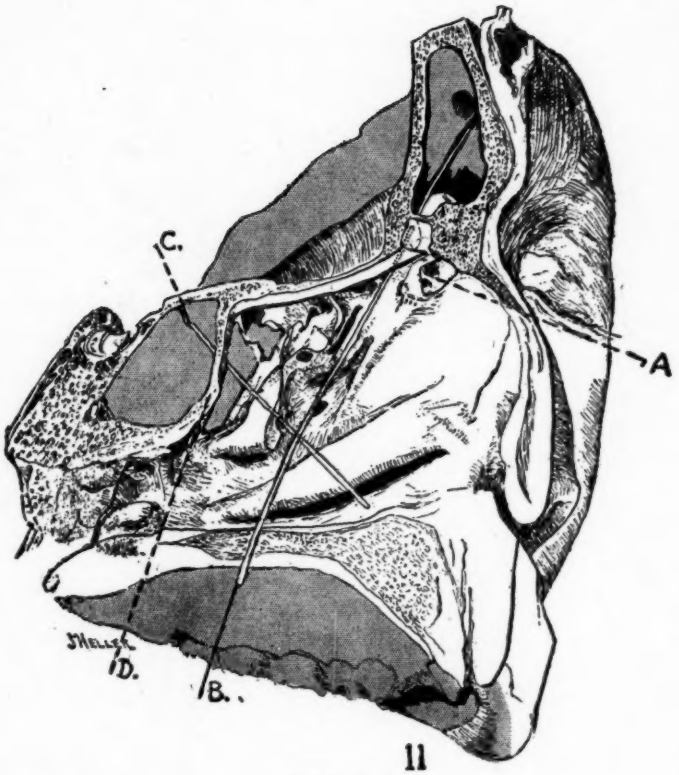

FIGURE 11.

Shows a three-quarter view of specimen with operation completed. The anterior half has been left "conservative"—i. e., the wall is intact, with all the openings free as far as one and one-half millimeters of the cribriform plate. An infundibular cell, A, has been opened, as shown first in Figure 5. The posterior half is "radical"—i. e., the anterior wall of the sphenoid cell has been removed with its entire postethmoidal part (lateral part, which is one-half or two-thirds of its extent). The postethmoidal cells have of necessity been dealt with radically to accomplish this—i. e., their entire nasal side has been removed. Bristle, B, is shown passing from the nose into the frontal sinus. Bristle, C, is shown passing through the postethmoidal-sphenoidal opening. A strip of bone, D, has been left to indicate the position of the sphenoidal-vomer junction

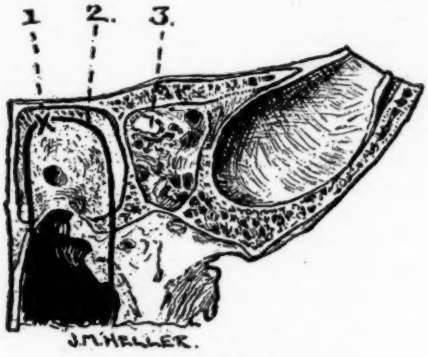

FIGURE 11A.

Shows diagrammatic representation of postethmoidal-sphenoidal cut shown in Figure 11. Transverse vertical section showing anterior sphenoidal wall viewed from behind. 1 the point X shows the entrance through the wall. The line 1-2 shows the extent of the incision. 3 the optic nerve.

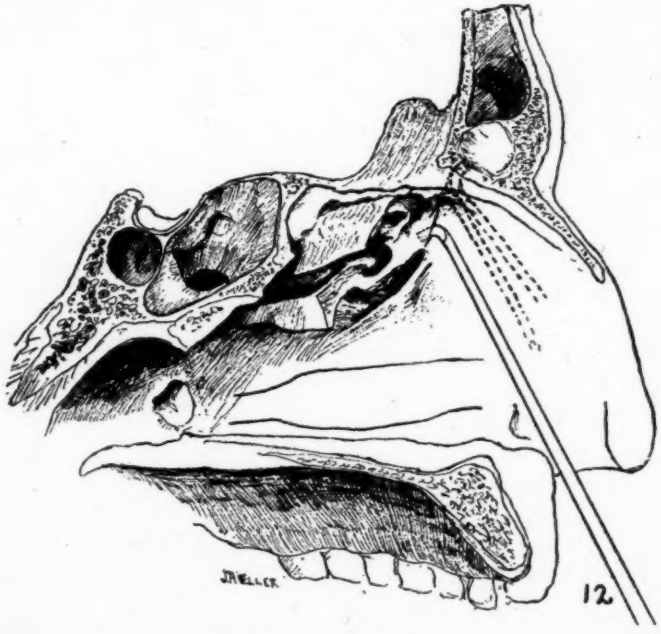

FIGURE 12.

Shows the knife in position for cutting away the uncinate process preparatory to Ingals' removal of the frontal sinus floor. Oftentimes the entire frontal floor may be cut away by the knife.

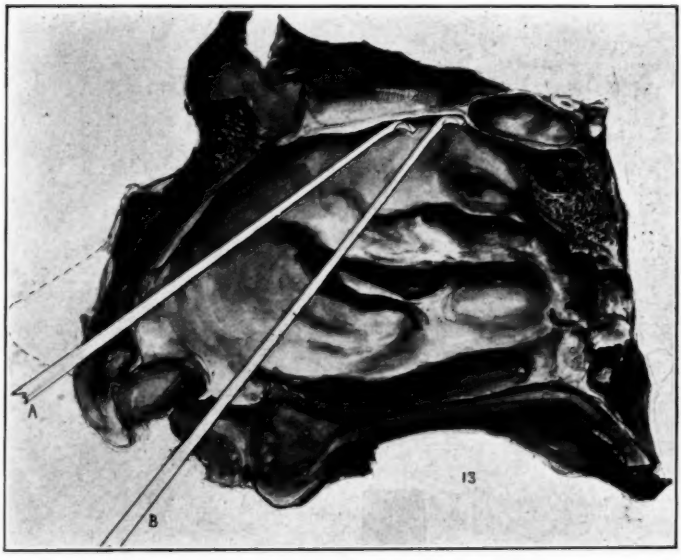

FIGURE 13.

Shows knife approaching a postethmoidal cell which lies on the top of the sphenoidal sinus and occupies about one-half of the body of the sphenoid. The line, A, shows position of approach. B shows the position for cutting. From considerable clinical experience I believe this cell would be opened by this technic.

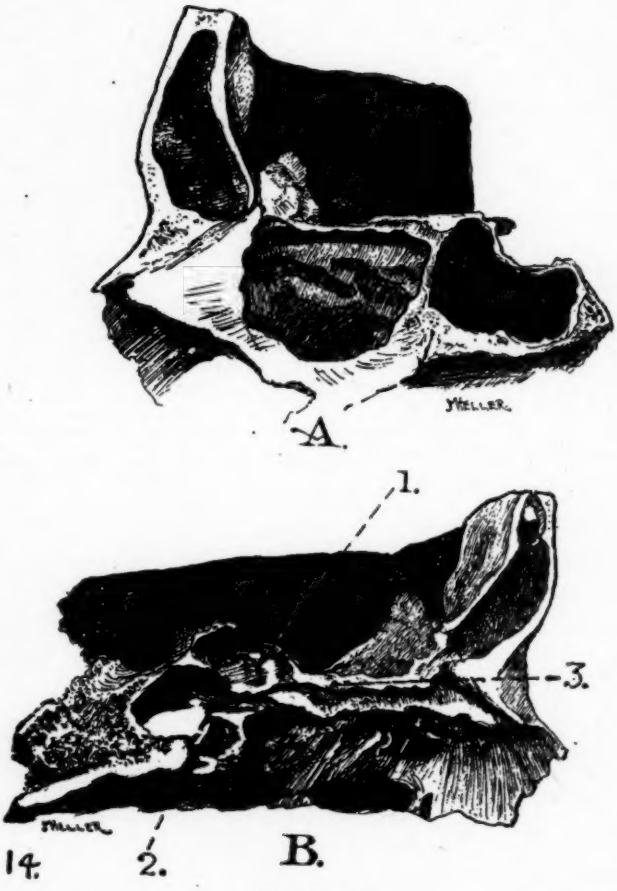

FIGURE 14.

A shows the usual sphenoidal sinus and the usual cribriform plate with their usual relations. B shows postethmoidal cell 1 placed altogether on top of sphenoidal sinus 2. Its lowermost limit is almost on the horizontal of the cribriform plate 3. This cell could not have been opened by this technic, nor, in my judgment, by any other extant.

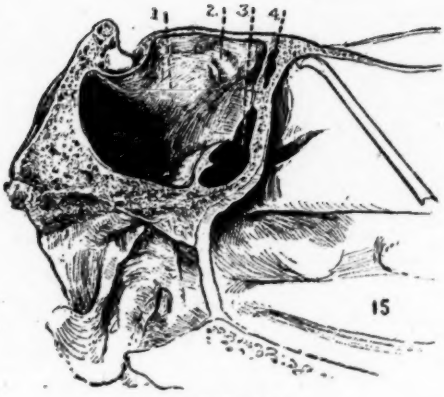

FIGURE 15.

Shows the left half of specimen. 1 right sphenoidal sinus extending into left side to border left optic canal 2. Left sphenodial sinus 3 also borders left optic canal. A postethmoidal cell, 4, also borders left optic canal. All three of these cells would probably have been opened by this technic operating on the left side.

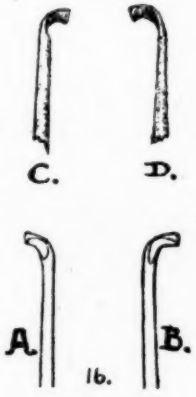

FIGURE 16.

A, sphenoid knife, B, turbinate knife. C and D, turbinate knives made right and left. Some of my confreres prefer right and left knives. I prefer the straight knife.

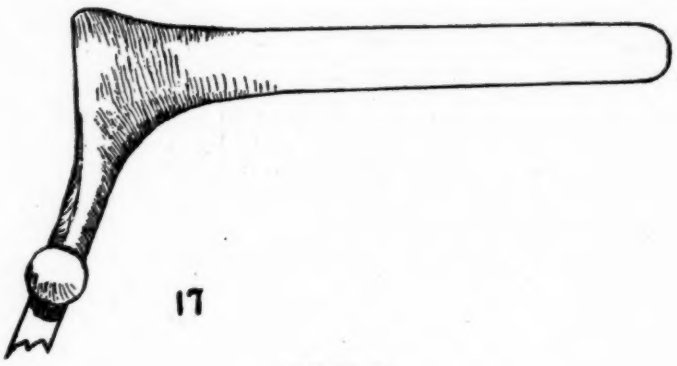

FIGURE 17.

Sphenoidal speculum. It is the primary bivalve nasal speculum made of tempered steel blades eighty-eight millimeters long, nine millimeters wide, knife edge thin near tip. It should have a set screw. It is a simple enlargement of the primary bivalve for this purpose, as Killian's enlargement is for the purposes of septum resection. It is thirteen millimeters longer and four millimeters wider than the usual model of Killian's. The sphenoidal knife used with this speculum is twenty-five millimeters longer than the regular knife. By means of this speculum the route to the sphenoid may be forcibly widened when it is too narrow.

XVIII.

ASTHMA ASSOCIATED WITH ETHMOIDAL DISEASE.*

By John Mackenzie Brown, M. D.,

Los Angeles.

The association of nasal diseases with asthma has been observed for many years, and especially the occurrence of polypi in these cases. There has been a great deal of work done recently in all departments of medicine, looking for local lesions which produce various chronic disorders throughout the body. It should be noted that the nasal lesions producing asthma are nearly all of ethmoidal origin, and that a more or less complete ethmoidal exenteration results in cure or in great improvement of the asthma.

According to Allbut, the pathologic anatomy of asthma is practically negative so far as the disease itself is concerned. Congestion, hyperplasia, and edema of the mucous membrane of the bronchi are frequently found, but he considers them either secondary to the repeated asthmatic attacks or due to an associated bronchitis. Williams and Longet first suggested that the actual spasm was produced by a contraction of the smooth muscle fibers which surround the bronchi. This was confirmed later by Hyde Salter in 1850, and worked out thoroughly by Brodie and Dixon in 1903. Allbut's explanation of the condition is the following: In moderate constriction of the bronchi, the powerful inspiratory efforts or movements are able to overcome the narrow tubes to a considerable degree. In expiration the forces which can be employed are much less efficient and are unable to expel the air, and the chest becomes fixed in a position of extreme distention. A

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, White Sulphur Springs, May 5, 1916.

very rapid swelling of the mucous and submucous tissues of the bronchi, due to vasomotor dilatation, was thought to be the cause of the asthmatic attacks, but it is now believed that the swelling of the mucous membrane is a secondary condition to the spasm of the bronchial musculature. The experiment of clamping the pulmonary vessels caused great engorgement of the bronchial mucous membrane, but did not produce any asthmatic symptoms. Horn, Freudenthal, and Keiper have observed in bronchoscopic examinations the presence of fibrous masses, ulcerations, and a general thickening of the mucous membrane. The accepted theory at present is that the symptoms are due to a spasm of the bronchial musculature, and that there undoubtedly is an edema of the mucous membrane, but that this is secondary. The question as to how this spasm is produced, whether by reflex or direct action on the muscle fibers by some substance in the circulation, has not been settled. A reflex necessitates action through the nervous system, while the direct action on the muscles could occur through the circulation, regardless of the nerves. The bronchial spasm has been attributed to the introduction of a foreign proteid to which a sensitization has been produced, or to a reflex irritation. Whether we accept the theory of hypersensitization or the reflex theory, there must be some other etiologic factor, because the same conditions in the nares present themselves in many cases, whereas only a comparative few develop asthma. Dundas Grant suggests that disturbance of the higher cerebral centers may interfere with the inhibition of reflex action in asthmatics. In our series of cases nearly all were of the neurotic type, and were so before the advent of any asthmatic symptoms.

The reflex theory implies that these attacks are brought about by an irritant in the nares or at some other part of the body, thereby causing a reflex stimulation of the smooth muscle of the smaller bronchi. The irritation in the nose causing reflex action could come from contact of the ethmoid with the septum, as this is the most sensitive area, or the altered secretion may be the irritant. Every case in our series had a contact of some kind in the nares. The sensory fibers of the nasal mucous membrane are supplied by the fifth nerve, and, according to Landois, the origin of the sensory roots of the

fifth nerve are connected with the motor roots of all the nerves arising in the medulla except the sixth nerve. Therefore, this connection accounts for the numerous and varied reflexes from irritation of certain branches of the fifth nerve. The tenth or vagus nerve arising from the medulla has intimate relationship with the nucleus of the fifth nerve, and supplies the motor fibers to the nonstriated muscles of the bronchi, or at least goes to form the anterior and pulmonary plexuses from which the nerves supplying these muscles originate.

The Anaphylactic Theory.—The experiments of Theobald Smith, Auer and Lewis, Otto, Meltzer, Matthews, Goodale, and others have shown that the injection of foreign proteids produce a sensitization of the body to that particular proteid, and that a subsequent injection of the same proteid produces the symptom complex known as anaphylactic shock, which has many characteristics of an attack of asthma. The phenomena exhibited in the bronchial tubes is undoubtedly, as the experiments of the above authors show, the same as in true asthma—i. e., spasmodic contraction of the involuntary muscles of the smaller bronchi. Just how the muscles are stimulated is unknown, whether it be due to stimulation by the toxin in the blood on this particular musculature, or by acting through the nervous system.

Auer and Lewis in their experiments obtained the same results in pitted as in unpitted animals, and the same symptoms of anaphylactic shock were observed after section of the vagi; these investigators will not, however, go so far as to state whether it is due to stimulation of the nerve terminals of the vagus or direct stimulation of the toxins on the musculature.

Matthews reports the same anaphylactic symptoms in animals in which the vagi had been severed and sufficient time had been allowed for complete degeneration of the peripheral fibers, as he obtained in animals in which the vagi had not been disturbed; he believes that the direct stimulation of the muscles through the blood is undoubtedly the cause of the spasm.

The investigations of Gay and Southard led them to believe the phenomena to be due to stimulation of the respiratory centers, causing violent inspiratory efforts of the diaphragm. The work of Auer and Lewis, and Matthews would seem to prove this to be erroneous.

Shultz has demonstrated that smooth muscle from a sensitized animal would contract more readily than from one non-sensitized.

In asthma due to pathologic lesions in the nose the absorption of pus or mucopus from these lesions is given as the cause of the attacks. Todd draws attention to the fact that sufferers from hay fever subsequently develop a distinct pathologic condition in the ethmoid cells; and that the asthmatic attacks in these cases may be due to inhaled pollen or to the ethmoidal diseases.

Koesler and Moody believe that bronchial asthma is due to a focal infection in the bronchi; that the subsequent bronchitis due to this infection is the primary cause of the asthma; and that the asthmatic patient has become sensitized to the infection causing the bronchitis. They found three anaerobic bacteria, two bacilli, and one streptococcus, in addition to the common aerobic bacteria. These bacteria, they believe, produce amines which cause the spasm of the muscles of the bronchi, and also cause ulcerated areas from which rapid absorption results.

In all our cases there was demonstrable some involvement of the ethmoids, and, no matter what other pathologic condition existed in the nose, this was a constant factor. Only five of our cases had suppurative ethmoiditis, and the probable reason that these cases do not result in asthma more frequently is that suppuration for any length of time will cause a degeneration of the normal mucous tissues and the nerve terminals.

There must be some underlying factor in these cases of ethmoiditis that will produce asthma, and this we believe to be the neurotic state of the individual, as we have all seen nares full of polypi with no asthmatic symptoms. We further believe that there must be asthmatic attacks due entirely to reflex action.

Mackenzie reports a case of a woman in whom he was able to produce repeated attacks of asthma by showing her an artificial rose. Chavonne reports a case of asthma of three years' duration due to a small papilloma of the uvula which was entirely cured by the removal of the tumor.

Two similar cases occurred in our practice. One, a woman whose asthma had been always relieved when she went to a

certain resort. While enjoying perfect health at this resort she received a letter containing bad news, and was immediately seized with a violent asthmatic attack lasting for several hours. The other is a case with ethmoiditis, in which an attack of asthma occurs instantly upon going from a warm room to one several degrees colder.

Landois, by irritating the nasal branches of sphenopalatine nerve, produced asthmatic symptoms, and Brodie and Dixon were able to produce typical asthma, after decerebration, by stimulation of the nasal mucous membrane. This reflex was abolished by section of the vagi; and even if the spasm had already commenced, it relaxed at once on section of the vagi. They also proved that the upper parts of the nares were the most sensitive to stimulation.

Whatever the theoretical explanation may be, two facts have been observed for a long time by rhinologists: first, that the association of chronic ethmoiditis and asthma is marked; second, that great improvement in asthmatic symptoms follows removal of pathologic nasal lesions.

Our desire in this paper is to report observations on twenty-seven asthmatic cases in which exenteration of the diseased ethmoid labyrinth was done. It is worth noting, also, that these patients were great sufferers, and many of them had gone through the list of asthma remedies without relief other than temporary.

The ages of the patients in this series range from four years to sixty-two years.

Two were under ten years.
One, ten to twenty years.
Six, twenty to thirty years.
Eight, thirty to forty years.
Five, forty to fifty years.
Four were over fifty years.
Females, fourteen; males, thirteen.

There was a marked deviation of the septum in thirteen cases. The antrum was involved on one or both sides in three cases, the frontal sinus in two cases, and the sphenoid in two cases. The ethmoids were involved in every case; twenty-two were of the hypertrophic type, with no demonstrable pus, and five were suppurative.

The treatment in this series consisted in as near a complete exenteration of the ethmoids as possible, according to the method of Mosher. The septum was only operated upon when it was necessary in order to do a satisfactory ethmoid exenteration, or when the deviation would still cause some irritation in the nose after the exenteration. After an exenteration the deviated septum seldom interfered with respiration or drainage from the sinuses. The antrum was treated by the Krause operation, and it was noticed in two antrum cases that treatment directed to the antrum first, greatly lessened the ethmoidal lesions and also the asthma. The frontal and sphenoidal sinuses were treated at the same time as the ethmoidal exenteration, by simple enlargement of the frontonasal duct or the sphenoidal opening. In children tonsils and adenoids were removed.

THE RESULTS OF THE OPERATION.

Six were completely relieved, having no return of symptoms to date, the periods in which no attacks have occurred ranging from three to eighteen months after operative interference. Twelve were decidedly improved—that is, having only slight attacks at intervals, and these mostly during acute attacks of rhinitis or other physical ailments. Seven were but slightly improved, and two were not improved at all. In the latter two classes six had marked bronchial pathologic lesions.

We are convinced from our observations on these patients that hypertrophic ethmoiditis is a distinct etiologic factor in the production of asthma. The complete exenteration of the ethmoids offers the best results in chronic cases. We realize the great difficulty in doing a complete exenteration, as it is practically impossible to remove all the mucous membrane in the labyrinth. A small portion of the diseased mucous membrane seems to be able to continue the attacks. In asthma due to nasal lesions, when there has been a marked bronchial condition, the bronchial lesions will continue the asthma in spite of any operative procedures in the nose.

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No.	Age	Duration	Nasal Findings	General Findings	Operation	Time of Observation	Results
1	45	10 years	Septum deflected, hypertrophic ethmoiditis with many polypi. No pus.	Marked bronchitis, emphysema	Ethmoid exenteration.	3 months	Attacks lessened in number and severity.
2	40	3 years	Chronic suppuration of the ethmoids and antrae.	Moderate bronchitis.	Ethmoid exenteration.	4 months	Attacks lessened in number and severity.
3	43	5 years	Hypertrophic ethmoiditis. Middle turbinates almost filled the nose. No pus.	Moderate bronchitis.	Ethmoid exenteration.	8 months	Attacks lessened in number and severity.
4	54	6 years	Hypertrophic ethmoiditis with many polypi.	Moderate bronchitis.	Ethmoid exenteration.	12 months	Attacks lessened in number and severity.
5	41	6 mo.	Hypertrophic ethmoiditis with right nares full of polypi. No pus.	Negative.	Ethmoid exenteration.	3 months	Attacks lessened in number and severity.
6	38	6 years	Very narrow nares, hypertrophic ethmoiditis. No pus.	Marked bronchitis, emphysema	Ethmoid exenteration.	6 months	Patient notices no difference in number or severity of attacks.
7	28	12 years	Septum deflected, hypertrophic ethmoiditis with many polypi in right nares. No pus.	Moderate bronchitis, emphysema	Septum resection and ethmoid exenteration.	8 months	Attacks lessened in number but not in severity.

8	39	20 years	Septum deflected, hypertrophic ethmoiditis with very large middle turbinates. No pus.	Marked bronchitis, emphysema	Septum resection, ethmoid exenteration.	14 months	Attacks lessened in number and severity. Patient was unable to do any work for four months prior to operation. Since operation has been able to do housework.
9	49	6 years	Septum deflected, chronic suppuration of the ethmoids, antrae and right frontal.	Moderate bronchitis.	Septum resection, Krause on the antrae, ethmoid exenteration and right frontonasal duct.	8 months	Decided lessening of number and severity of attacks. Patient was unable to do any work prior to operation, but is now able to work half the time.
10	26	6 years	Septum deflected, hypertrophic ethmoiditis with many polypi. No pus.	Marked bronchitis, emphysema	Septum resected, ethmoid exenteration.	18 months	Decided lessening in number and severity of attacks.
11	30	2 years	Septum deflected, hypertrophic ethmoiditis. No pus.	Slight bronchitis.	Septum resection, ethmoid exenteration.	18 months	Attacks gradually became less and entirely ceased nine months ago.
12	27	2 years	Hypertrophic ethmoiditis with many polypi. No pus.	Slight bronchitis.	Ethmoid exenteration.	10 months	The attacks entirely disappeared six weeks after operation.
13	29	7 years	Hypertrophic ethmoiditis, many polypi, very narrow nares. No pus.	Chronic bronchitis, emphysema, chronic pulmonary tuberculosis.	Ethmoid exenteration.	14 months	Decided lessening in number and severity of attacks.

No.	Age	Duration	Nasal Findings	General Findings	Operation	Time of Observation	Results
14	38	8 years	Hypertrophic ethmoiditis. No pus. ch	Moderate bronchitis.	Middle turbinates removed 3 years ago, ethmoid exenteration two years later.	3 years	After removal of middle turbinates, patient was completely relieved for two years, when asthma again developed. After exenteration, no attacks.
15	13	6 years	Deviated septum, hypertrophic ethmoiditis, middle turbinates, hypertrophic tonsils and large adenoids. No pus. ch	Moderate bronchitis.	Septum resection, tonsils and adenoids removed.	6 months	No asthma since operation.
16	62	17 years	Septum deflected, hypertrophic ethmoiditis with polypi. Slight pus discharge from both nares. After ethmoid operation pus was discovered in right sphenoid. ch	Marked bronchitis, emphysema	Septum resection, ethmoid exenteration.	4 years	Attacks decidedly lessened in number and severity.
17	53	6 years	Hypertrophic ethmoiditis. No pus. ch	Marked bronchitis, emphysema	Ethmoid exenteration.	10 months	Attacks lessened in number and severity.
18	35	3 years	Hypertrophic ethmoiditis with many polypi. No pus. ch	Slight bronchitis.	Ethmoid exenteration.	10 months	No attacks since operation.
19	52	5 years	Septum deflected, hypertrophic ethmoiditis. ch	Slight bronchitis.	Right ethmoid exenteration.	5 months	No attacks since operation.
20	30	3 years	Septum deflected, hypertrophic ethmoiditis. No pus. ch	Slight bronchitis.	Septum resection, ethmoid exenteration.	8 months	Decided lessening in attacks in number and severity.

21	30	1 year	Suppuration of ethmoids, antrae, left frontal and left sphenoid.	Negative.	Ethmoid exenteration, enlargement of fronto-nasal duct and sphenoidal opening. Krause on antrae.	11 months.	Patient has had one attack since operation, due to an acute rhinitis, and several mild attacks after exposure to severe weather.
22	35	4 years	Hypertrophic ethmoiditis with polypi. No pus.	Slight bronchitis.	Ethmoid exenteration.	22 months	There has been no attacks for the past fourteen months.
23	20	3 years	Septum deflected, hypertrophic ethmoiditis with very large middle turbinates. No pus.	Slight bronchitis.	Septum resection, ethmoid exenteration.	12 months	No asthma since operation.
24	4	1 year	Hypertrophic ethmoiditis, hypertrophy of tonsils, and large adenoid.	Negative.	Adenoid and tonsils removed.	15 months	Attacks lessened in number and severity.
25	4½	1½ years	Hypertrophic ethmoiditis, hypertrophy of tonsils, and large adenoid.	Negative.	Adenoid and tonsils removed.	9 months	No asthma since operation.
26	28	7 years	Septum deflected, hypertrophic ethmoiditis. No pus.	Slight bronchitis.	Ethmoid exenteration.	10 months	No asthma since operation.
27	25	2 years	Septum deflected, hypertrophic ethmoiditis. No pus.	Slight bronchitis.	Septum resection, ethmoid exenteration.	9 months	Attacks lessened in number and severity. Slight attacks during acute rhinitis, and whenever he has digestive disturbances.

No.	Age	Duration	Nasal Findings	General Findings	Operation	Time of Observation	Results
14	38	8 years	Hypertrophic ethmoiditis. No pus.	Moderate bronchitis.	Middle turbinates removed 3 years ago, ethmoid exenteration two years later.	3 years	After removal of middle turbinates, patient was completely relieved for two years, when asthma again developed. After exenteration, no attacks.
15	13	6 years	Deviated septum, hypertrophic ethmoiditis, middle turbinates, hypertrophic tonsils and large adenoids. No pus.	Moderate bronchitis.	Septum resection, tonsils and adenoids removed.	6 months	No asthma since operation.
16	62	17 years	Septum deflected, hypertrophic ethmoiditis with polypi. Slight pus discharge from both nares. After ethmoid operation pus was discovered in right sphenoid.	Marked bronchitis, emphysema	Septum resection, ethmoid exenteration.	4 years	Attacks decidedly lessened in number and severity.
17	53	6 years	Hypertrophic ethmoiditis. No pus.	Marked bronchitis, emphysema	Ethmoid exenteration.	10 months	Attacks lessened in number and severity.
18	35	3 years	Hypertrophic ethmoiditis with many polypi. No pus.	Slight bronchitis.	Ethmoid exenteration.	10 months	No attacks since operation.
19	52	5 years	Septum deflected, hypertrophic ethmoiditis.	Slight bronchitis.	Right ethmoid exenteration.	5 months	No attacks since operation.
20	30	3 years	Septum deflected, hypertrophic ethmoiditis. No pus.	Slight bronchitis.	Septum resection, ethmoid exenteration.	8 months	Decided lessening in attacks in number and severity.

21	30	1 year	Suppuration of ethmoids, antrae, left frontal and left sphenoid.	Negative.	Ethmoid exenteration, enlargement of frontonasal duct and sphenoidal opening. Krause on antrae.	11 months.	Patient has had one attack since operation, due to an acute rhinitis, and several mild attacks after exposure to severe weather.
22	35	4 years	Hypertrophic ethmoiditis with polypi. No pus.	Slight bronchitis.	Ethmoid exenteration.	22 months	There has been no attacks for the past fourteen months.
23	20	3 years	Septum deflected, hypertrophic ethmoiditis with very large middle turbinates. No pus.	Slight bronchitis.	Septum resection, ethmoid exenteration.	12 months	No asthma since operation.
24	4	1 year	Hypertrophic ethmoiditis, hypertrophy of tonsils, and large adenoid.	Negative.	Adenoid and tonsils removed.	15 months	Attacks lessened in number and severity.
25	4½	1½ years	Hypertrophic ethmoiditis, hypertrophy of tonsils, and large adenoid.	Negative.	Adenoid and tonsils removed.	9 months	No asthma since operation.
26	28	7 years	Septum deflected, hypertrophic ethmoiditis. No pus.	Slight bronchitis.	Ethmoid exenteration.	10 months	No asthma since operation.
27	25	2 years	Septum deflected, hypertrophic ethmoiditis. No pus.	Slight bronchitis.	Septum resection, ethmoid exenteration.	9 months	Attacks lessened in number and severity. Slight attacks during acute rhinitis, and whenever he has digestive disturbances.

XIX.

REPORT OF THREE CASES OF BRAIN ABSCESS OF OTITIC ORIGIN.*

By George M. Coates, M. D.,

Philadelphia.

Case 1.—M. O., aged fifty-one years, a waiter, came to the Out-Patient Department of the Pennsylvania Hospital, July 6, 1912, complaining of pain in the right ear of two days' duration. He gave a history of having had a right sided aural discharge for several days four months previously, but with no discomfort in the interim. Examination showed a bulging membrane, which was at once incised in the posterior portion of the membrana, a somewhat scanty discharge resulting. Some mastoid tenderness was present which became slightly more marked in the next few days, and he was admitted to the ward on July 9th. He did not complain particularly of headache. No signs of meningitis were present, and the leucocyte count was practically normal. The patient was a well developed and well nourished man. Eyes showed no spontaneous nystagmus, pupils were equal and reacted to light and accommodation, eye grounds negative. There was no aphasia. The teeth were in bad condition; throat normal and tongue slightly coated. Heart, lungs and extremities normal. Urine normal. Temperature, 99°; pulse, 88; respirations slightly high.

Expectant treatment was carried out for a few days, as the temperature continued low, but some slight swelling appearing over the mastoid, he was operated upon on July 15th. The mastoid cells were all found normal with the exception of the antrum, the tegmen of which appeared soft, if not necrotic,

*Read at the meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, held at Philadelphia, March 3, 1917.

this softened area extending outward into the temporal ridge. The dura was exposed for inspection and appeared to be under considerable pressure, so it was incised and the brain searched with a thin knife blade. In a direction inward and upward, about an inch from the surface, an abscess cavity containing about two drams of pus was encountered. It appeared to have rather firm walls. Gentle washing cleaned out the pus; a cigarette drain was inserted, and the lower portion of the skin wound closed. Cultures from the pus failed to show any growth. Recovery was slow but perfectly uneventful, the abscess continuing to drain nicely, although the middle ear ceased at once to discharge. The drain was removed in about ten days and the wound allowed to close by granulation, which it did completley in about six weeks. During this time the temperature never rose above 99 4/5°, being normal for the most part, the pulse ranging from 64 to 88. Respirations were always a little rapid.

This case showed nothing clinically to suggest brain abscess, the bulging of the brain and dura through the operative bone wound being the only indication which led to its discovery. As the contained pus was apparently sterile, and as the cavity had definite walls, it is probable that it had its inception at the time of the first ear trouble, four months earlier. It illustrates very well the type of abscess that is favorable to recovery, namely, a walled-off, chronic process with sterile pus, or at least having organisms of low vitality, discovered at the time of operation without having given rise to pressure symptoms.

Case 2.—C. C., twelve years old, school boy, admitted to the Pennsylvania Hospital May 24, 1913. Temperature, 99°; pulse, 100; respirations, 24. He had had a simple mastoidectomy on the right side two years before, followed by a radical on the left side one year later. On admission he gave a history of pain in the right ear for five days, accompanied by a free discharge of pus. For two days past there had been marked general headache. He was a frail, undersized boy, irrational, whining, with considerable photophobia. The eyes reacted well to light and accommodation; grounds apparently normal. Heart and lungs were negative. The neck was quite rigid, suggestive Kernig signs were present on both sides

and a doubtful Babinski; reflexes not markedly increased. There was swelling and redness over the right mastoid region, which was markedly tender. The right membrana showed a large central perforation in a thickened drum, freely discharging foul pus with a distinct musty odor. The left ear, on which a radical operation had been performed one year before, was not entirely dry. The leucocyte count was 21,700, with a high percentage of polymorphonuclears. By lumbar puncture the spinal fluid was found to be cloudy and under considerable tension, with the polymorphonuclears sixty-seven per cent. No tubercle bacilli were found, and no growth was obtained on culture media.

Although all his symptoms were considerably relieved by the lumbar puncture, he was operated upon by Dr. Francis R. Packard, on whose service he was, and much destruction of bone in the upper portion of the mastoid found, extending into the temporal ridge and zygomatic cells. The dura was freely exposed and the wound packed with iodoform gauze. Whereas he had been markedly restless, delirious and noisy previous to operation, he now became much quieter and mentally more clear. Very foul pus continued to drain from the wound, and at this stage Dr. Packard went off duty and he came into my hands. The improvement following the first operation was not permanent, however, and during the next few days all of his former symptoms returned in greater force, the neck being well arched backward and his delirium increasing. On July 9th, ten days after the first operation, in dressing the wound, I noticed that the dura was tense and bulging in the bony opening through the site of the temporal ridge, where the removed bone covered an area about three by four centimeters. A paralysis of the external rectus muscle of the right eye was now apparent. The pus was very foul in odor. With very light anesthesia, I incised the dura and searched the underlying brain with a grooved director, when a large abscess cavity was discovered containing about an ounce of thin, foul pus. A rubber drain was inserted without washing and the wound dressed. The patient was very weak and partly comatose. The pus showed the colon bacillus in pure culture, and continued to drain very freely, and always foul, until, gradually sinking, he died five days later. At this

time there was considerable protrusion of the brain tissue into the wound, the protruded portion being necrotic. The temperature had ranged from 99° on admission to 104° on May 26th, where it stayed for two days. It dropped to 99° for a few days and again rose to 104 3/5° when the second operation was performed. Thereafter 103° was the lowest temperature until death. The pulse rate was commensurate with the temperature, running from 100 on admission to 160 shortly before death. Respirations were always rapid—from 24 to 32, and 52 at death. No autopsy was obtainable, but from the appearances presented, I think it probable that there was a second abscess that was not evacuated. The patient's condition while upon the operating table was so grave that I did not feel inclined to search further after draining the abscess.

Case 3.—Laura H., thirty years old, stenographer, was admitted to the Pennsylvania Hospital July 6, 1916, complaining of pain in the left ear, left sided headache and general malaise. Temperature, 102°; pulse, 92, and respirations, 24. Family history was without interest. She had had measles in childhood, which did not affect the ears as far as she could remember; had always had "catarrh," indigestion and constipation. The left ear had suppurated, with severe earache, fifteen years previously, and had ceased discharging after a few days. During the next few years she had had several similar attacks in the left ear, and finally the discharge had been almost continuous for the past six years, although always scanty. She had not had the ear treated, considering it a negligible matter, except for the impaired hearing.

The present illness dated back six days, when she developed an acute rhinitis from sitting in front of an electric fan while at work. Earache immediately followed and she went to bed. There was an attack of vomiting the same evening, and as she felt badly she was admitted to another hospital the next day, July 4th. Not getting any better here, and having almost continuous nausea and vomiting, she left this institution and was admitted to the Pennsylvania Hospital, where the drum was incised at once. Temperature on admission was 102°; pulse, 92; respirations, 24. She was a well developed and well nourished, full faced, rather pale adult, complaining bitterly of pain in the left ear. There was no adenopathy, the

eyes were slightly prominent, pupils widely dilated, equal, reacted normally to light, grounds normal. The nasal septum was deviated to the left, the teeth in fair condition, tongue coated, throat negative. A scanty discharge came from the left ear, and there was no retraction of the head, although slight pain on extreme flexion of the neck and a suggestion of Kernig's sign in the right leg. Reflexes normal. No spontaneous nystagmus was present, vestibular reaction to the caloric test normal. Widal and Wassermann reactions negative. The blood count showed a leucocytosis of 14,000, polymorphonuclears seventy per cent. Urine negative. Patient was very uncomfortable. No aphasia—mentally slightly sluggish.

On July 8th complained of considerable pain in the ear and back of the head, as well as in the nose. There was very little aural discharge. The eye examination by Dr. Wm. T. Shoemaker follows: Externally nothing noticeably abnormal. Pupils large and equal. Irides react to light. Ocular movements full. Eye grounds: media clear, discs well defined, vessels good, no hemorrhage or exudation or any evidence of intracranial trouble. Temperature, 99°; pulse, 80; respirations, 20. Her condition continued no worse for two days, when the temperature rose to 102°, pulse 84, respirations 20, and she vomited a grassy green substance. Pain was more intense on the vertex and occiput; there was slight rigidity of the neck, and Kernig's sign was positive in the right leg. Dr. Morris J. Lewis, the internist, confirmed the diagnosis of meningitis. The mastoid was not markedly tender, and there was no sagging of the posterosuperior canal wall. By lumbar puncture thirty-five cubic centimeters of fluid under pressure were withdrawn. Fluid was cloudy, there was slight reduction of Fehling's solution, cytologic count was one hundred and eighty-six, polymorphonuclears eighty per cent. The blood culture was negative. As the patient was much more comfortable following this, it was repeated on the following day, July 11th, and twenty cubic centimeters of cloudy fluid, not under pressure, were withdrawn, with apparently increased comfort. At midnight, however, she again complained of great pain, which morphin failed to control. She became weak and cyanotic during the remainder of the night and early morn-

ing. The pulse was very weak and the temperature went to 104 1/5°; respirations, 24. The report of the X-ray picture, taken July 10th, proved to be very misleading, as follows: "Extensive necrosis of the entire cell area of the left mastoid, sinus probably uncovered. No further evidence as to pressure or location of any suspected brain abscess."

The left mastoid was opened July 12th and found to be eburnated, without cells or necrosis, solid and intact; the kind frequently seen accompanying prolonged middle ear suppuration. Believing that we were dealing with a case of purulent meningitis with the possibility of brain abscess, I freely exposed and incised the dura of the temporal lobe, obtaining a little cloudy cerebrospinal fluid. The tension of the brain appeared to me to be increased, so I searched it cautiously with a grooved director and tapped an abscess cavity containing nearly an ounce of thick pus. Up to this time the patient's condition had been precarious. The temperature was 102°; pulse, 80, and respirations, 28, just before being placed on the table; she was comatose, cyanotic and breathing was slightly stertorous. The pulse was very weak. Very little ether was used in anesthetization. After draining the abscess, almost immediately her pulse improved, her color became normal, and her breathing was easier. A rubber drain was inserted, the wound packed and dressed, and she was sent to the recovery ward, by this time already conscious and apparently rational. Before I had finished dressing, however, word came that she was sinking rapidly, and when I reached her a moment later she was cyanotic, although the pulse was fair and continued so for several moments. Respiration became weak and ceased. Artificial respiration and stimulants were of no avail, and she died of respiratory failure.

The autopsy diagnosis was multiple (two) brain abscess in the left temporal lobe, purulent meningitis. The complete autopsy report follows:

"There is an operation wound with a drain inserted reaching into the cranial cavity behind the left ear. There is a slight clouding beneath the pia. The various arachnoid spaces contain cloudy fluid. The fluid in both ventricles is purulent—less so in the right. The right cerebrum is otherwise normal. The drain reaches into an abscess cavity within the temporal

lobe, about three centimeters posterior to its anterior end and opening in about the line of the inferior temporal sulcus. This cavity is just beneath the surface, measures about two centimeters in diameter, contains some purulent fluid and has a soft, thin wall of necrotic cerebral tissue. There is also another abscess which opens into the posterior descending horn of the left ventricle and to the surface proximal to the cerebellum at about the posterior third of the collateral fissure and slightly lateral to that in the fusiform gyrus. This abscess cavity is the larger of the two, in that its openings in both directions are large. There is about one centimeter of cerebral tissue in between the two cavities.

Microscopic Examination.—The edge of the brain cavity is densely infiltrated with leucocytes and to a less extent with plasma cells. There is no fibrous reaction about the inflammatory area. The cavity is not of long standing."

From the autopsy report these abscess cavities seem to have been of recent formation, and there is nothing in the history to indicate prolonged intracranial involvement except the duration of the aural discharge—fifteen years. Of course, we know that a brain abscess frequently lies dormant for long periods of time to eventually become active and dangerous under some fresh stimulation, such as the chilling from the electric fan and consequent aural reinfection in this case. Although we suspected brain abscess almost from the start from the character of the headache, nothing confirmatory of this could be discovered, and the development of the meningitis was so evident, that that exclusive diagnosis was made. I could not be quite content with this, however, or feel that it accounted for all of her symptoms, and therefore searched the brain at the time of operation, with the discovery of the first abscess. I did not suspect the second one. Immediately following the evacuation of the first abscess her condition was in all respects so satisfactory that I feel I was justified in believing the source of the trouble had been discovered and eradicated, and I sent her away from the table feeling that recovery was not at all to be despaired of. Whether the sudden collapse that followed within ten minutes was due to the rupture of the deeper abscess into the ventricle I do not, of course, know, but think that is probably what occurred, since

such a rupture was discovered at autopsy. Or it is possible that there had been a slow leakage there which accounted for the infection of the cerebrospinal fluid. There was no "stalk" or ascertainable avenue of infection to or from the middle ear, although this must undoubtedly have been the origin of the brain lesion.

These three cases only serve to emphasize still more the well established dictum that a chronic discharging ear, no matter how trivial it may appear, or how quiescent during long periods of time, is always a serious menace to the life of the patient. No one fact pertaining to suppurative otitis media is, perhaps, better established, and yet how many such middle ear conditions are neglected and temporized with. J. W. Murphy lays stress upon this point in his article read before the society in June, 1915, and considers cerebral abscess of otitic origin almost always an acute process, and that, therefore, eye pressure symptoms have not time to develop, as in the more slowly growing tumors. In only one of my cases was there any paralysis apparent—that of one of the eye muscles in the second case—and this came on late in the disease. Neither were the mental symptoms more conclusive or helpful in establishing the diagnosis. In one of these cases, the one that recovered, there was little elevation of temperature, but in the other two the febrile reaction was marked—none was ever subnormal. The pulse rate was rapid, at least commensurate with the temperature, as were also the respirations. With wakefulness and delirium rather than slow cerebration, and mental alertness instead of drowsiness, the absence of eye pressure symptoms or of paralysis, and in two of the three cases a concomitant meningitis existing, the diagnosis of a brain abscess was a difficult task. As a matter of fact, it was only made on the operating table, after a search of the brain on suspicion, in two of the cases.

XX.

SEVERE EPISTAXIS IN A WOMAN WITH DIABETES, MYOMATA OF THE UTERUS AND ACUTE MASTOIDITIS.*

By Wolff Freudenthal, M. D.,

New York.

On account of its numerous complications, the following case may be of interest to the Fellows of this section:

Mrs. ———, aged about forty-five years, called on me in the middle of the night of January 16, 1917. She was accompanied by two physicians, who had been unable to stop the severe epistaxis from which she had suffered for several hours. The bleeding had started in a mild form at noon; a physician was called in and left a prescription for adrenalin with instructions; but the hemorrhage continued and became profuse during the evening, when the two other colleagues were summoned. When I saw the patient she showed some signs of acute anemia. With some difficulty the bleeding was finally stopped by plugging the nose anteriorly. Adrenalin, it was soon found out, had here the opposite effect to what it generally has—it increased the hemorrhage.

As this lady was of an age when menopause sometimes occurs, and having encountered numerous instances of vicarious menstruation, I inquired about the menses, and was told that she was just having them, that they occurred every three weeks and lasted for eight days or more. Her family physician, furthermore, stated that she was suffering from multiple myomata of the uterus, the removal of which before the next period I strongly advocated, in order to avoid further trouble from the nose.

The patient was taken home and rest and ice applications

*Read before the New York Academy of Medicine, Section on Laryngology, March 28, 1917.

ordered. The next afternoon I visited her and removed the gauze, but had to pack again on account of the occurrence of quite some bleeding. During the following night a severe hemorrhage set in, which was arrested by my assistant. The next morning (January 18th) she complained of toothache, having had for some time past several carious teeth, one of which had to be extracted a week before. Undoubtedly the toothache was caused by the ice applications, which now had to be discontinued, this complicating somewhat the treatment of the epistaxis. Besides, the question of her having another tooth extracted came up, but it was deemed inadvisable to do so on account of the continued epistaxis, which, I believed, would not cease until her menses were over. Such was the case, for the bleeding stopped entirely on January 22d. Before that happened, however, an otitis media set in on January 19th, so that a paracentesis had to be made, with relief of a good deal of the pain from that source.

January 25th, on examining the nose and throat, I was struck by the peculiar aspect of the pharynx. The nose did not present any peculiar feature except that the mucosa was very dry, and anteriorly on the septum, at the socalled locus Kiesselbachii, there was the place from which the bleeding had started. The pharynx, however, was different. The entire mucous membrane was also very dry. The velum and its vicinity was somewhat edematous, and on the wall of the pharynx there was seen a distinct superficial ulceration. No lues nor tuberculosis being present, I had the urine examined, and it was found to contain four and four-tenths per cent of sugar plus acetone.

Naturally that upset our plans as to the removal of the myomata of the uterus, and it was thought best to resort to applications of Roentgen rays instead, which, by the way, had given very good results so far. In our specialty, it seems to me, we ought to make more use of those rays than we do. But that only apropos.

As soon as the patient was able, two teeth were extracted under cocain and the dental neuralgia arrested.

In the meanwhile, however, her otitis media had commenced to trouble her. A profuse purulent discharge, pain and swelling over the mastoid—in short, positive signs of a mastoid

involvement—appeared. But I hesitated to operate, as the sugar had gone up to five per cent and the patient was in nowise a good surgical risk. As soon as she was somewhat stronger, and when the sugar had come down to three per cent, I found it necessary to operate, being well aware of the chances I took. The result was good beyond our expectation. She stood the narcosis (gas oxygen) very well, and healing of the wound was uneventful.

In going over this case it will be noted that there are two points of special interest. The first is that the possibility of diabetes being present was first suggested by a laryngologist. When I had a chance to examine the pharynx thoroughly, its peculiar appearance made me suspect diabetes. Years ago I described diabetic ulcerations of the pharynx and larynx,[1] but, with few exceptions, my reports were not noticed. Ever since I had occasion to see such cases occasionally, but did not collect them for publication, in the belief that the occurrence of such lesions had been established firmly by me.

Lately, Arrowsmith[2] published the brief history of a case of diffuse diabetic ulcerations of the pharynx and larynx, which would make, as he adds, "the seventh case of diabetic ulceration of the upper air passages so far recorded—the sixth, unless further search reveals Westbrook's observation." I too have searched for Westbrook's publication, but have been unable to find a single mention of it in the literature, either before or after my publication (sc. of five cases). The only one who had noticed something similar, namely, an abrasion on a vocal cord in a diabetic patient, was Edward Law of London. But that was only a side issue, to which he paid no attention. The main feature in his case was a subglottic nodule of doubtful origin.[3]

When I first described such cases, I thought to be justified in drawing the distinction between benign and malignant ulcerations. I am still of the same opinion. Whether the character of the ulcer depends upon the underlying disease, diabetes, as seems natural to suppose, or upon local conditions, is impossible to state. Our patient certainly belongs to the class that heals easily under general and local treatment. I am almost inclined to believe that in some, perhaps in many, instances the ulcers heal without any local applications. Producing no

symptoms (especially no dysphagia), they are so often overlooked.

The second point of interest in my case was that a mastoid operation had to be done on a patient affected with diabetes. That such a complication is a grave one, especially in a subject still suffering from an acute anemia, nobody can deny. Such an operation is a far more serious affair than ordinarily, and any surgical intervention under such circumstances may have a fatal outcome. Still, no other choice was left than to open the mastoid and give free exit to the pus collection. It may be added that the preoperative mental condition of the patient was all that could be desired. She did not know that she was suffering from diabetes, nor from a mastoid. We told her that an abscess had to be opened, and she did not inquire for more details. I consider that psychical condition a very important factor in major operations, and the more we learn to act accordingly, the better it will be for the patient. As to the operation, no stress was laid on elegance nor thoroughness; free drainage had to be established in as short a time as possible. And that was done. The patient slept almost constantly for twenty-four hours after the operation; one-fourth grain of morphin had been given before. The final outcome was extremely gratifying.

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XXI.

SEPTAL HEMORRHAGE—ITS CURE BY SUBMUCOUS ELEVATION.

By John Leshure, M. D.,

New York.

The steps of this procedure are:

1. Induction of analgesoischemia (this term is suggested for the state which follows the local application of cocain and epinephrin or their substitutes, and its adoption is urged as being more comprehensive than the terms generally in use).

2. Incision and elevation of the septal mucoperichondrium backward for one inch and down to the floor of the nose.

3. Compression of the elevated membrane between the blades of a special forceps.

4. Replacement of the flap and packing in the usual manner for from twelve to twenty-four hours.

Indications for operating:

1. Cases of septal hemorrhage which resist ordinary methods of treatment, such as cauterization, astringents, etc., and those in which no bleeding point can be readily found.

2. Cases of chronic crusting of the septum accompanied by bleeding.

3. Cases of septal hemorrhage of mild or severe degree in children who will not tolerate local treatment and who are best operated upon under general anesthesia.

The principle of cure is obviously the destruction of the thin walled superficially placed vessels which bleed at the slightest provocation, and the ability to grasp the vessel between the blades of a forceps constitutes a distinct advantage, since pressure crushes the intima coat of the vessel, and obliteration of its lumen is thus accomplished.

The bleeding points are usually low down and anterior, consequently the incision should be placed as far forward as possible, and the elevation carried well down to the nasal floor.

If it is found difficult to reach with the compression forceps the vessels near the floor of the nose, a horizontal incision should be made along the floor joining the lower end of the vertical incision, or, in other words, one makes an L-shaped flap, all parts of which can readily be compressed. These two incisions sever the continuity of the vessels passing through Stenson's canal and those which pass into the mucous membrane from the maxillary crest.

Some of the vessels are of considerable size, and their anastomosis is so free that cauterization often fails to accomplish a cure.

In cases of chronic crusting accompanied by bleeding, the membrane is bound down by fibrous bands which interfere with the circulation and cause distention of the vessels at one point and constriction at another.

When these bands are divided by the elevation the blood supply readjusts itself, becoming more nearly normal, and the general tone of the membrane is decidedly improved, with a lessened tendency toward crusting.

This improvement has been noted after the submucous resection, both by the writer and others, and will be referred to later.

It is easier to operate between attacks, but if other means fail, one can usually check the bleeding in the following manner and operate immediately:

A pledget of gauze soaked in a ten per cent solution of cocain in 1:1000 adrenalin is placed in the bleeding nostril while the opposite septal surface is painted with cocain-adrenalin syrup (fifty-five per cent strength), the object being to secure a reflex vasomotor constriction of all the neighboring vessels.

If oozing still continues, the bleeding side may be packed firmly with oily gauze, which will allow but little blood to escape, and while still in situ, incision and elevation underneath the packing may be carried out. Unless the case is one of marked hemophilia, no further difficulty will be encountered.

Overaction of the heart, due to extreme nervousness, is apt to cause continued hemorrhage, and a hypodermic of morphin and adrenalin in proper proportions will check the bleeding and quiet the patient's fears.

A good balance is one-sixth grain of morphin sulphate dis-

solved in ten minims of 1:1000 solution of adrenalin. If more adrenalin is used, the morphin should be increased to prevent excessive rise in blood pressure.

Morphin dilates the medium sized vessels, at the same time slowing the action of the heart, thus lowering general blood pressure.

Adrenalin increases the force and frequency of the heart beat, and this action, added to the contraction of the peripheral vessels, raises the pressure.

By giving the drugs together the desired effect of both is obtained—viz., lowered blood pressure and contraction of all of the small blood vessels—owing to the predominant action of the morphin.

Children and nervous adults may be given a general anesthetic if necessary, and the anesthesia may be limited to the primary stage if the septum is painted with cocain-adrenalin syrup.

Shock and bleeding are considerably lessened when local analgesoischemia is added to the general anesthesia.

Ligation of the bleeding vessels has been suggested by some writers, but the membrane is usually so friable that such a proceeding must be attended with considerable difficulty.

A smooth flat-bladed angular forceps has proved most convenient for compressing the membrane, but an ordinary dressing forceps answers almost equally well. Care should be taken not to use sufficient pressure to cause necrosis and sloughing.

The wound edges may be brought together with sutures if one cares to spend the time upon this refinement of technic. Usually healing occurs promptly upon replacement of the flap, and in the writer's series there has been no instance of perforation or other untoward result.

A convenient method of packing is with strips of oily gauze introduced in successive layers with a piece of rubber tissue placed next to the wound.

If packed in this manner the strips may be removed in reverse order and without danger of disturbing the wound edges.

The after-treatment is that usually given resection cases—simple cleansing and protection of the wound with boric ointment or some similar preparation.

Dr. J. A. Thompson[1] and Dr. Joseph Prenn[2] both cite cases of septal hemorrhage cured by submucous resection of the septum.

The writer's experience, however, based upon forty-one cases of submucous elevation with compression, leads him to believe that resection of the cartilage is unnecessary, unless, of course, indicated because of nasal obstruction, or for other reasons; in other words, elevation and compression of the mucoperichondrium will cure practically all cases of septal hemorrhage.

In the opinion of many rhinologists, resection of the septum should not be undertaken when the patient is less than sixteen years of age, and since many of our cases of epistaxis occur in children, a method which will not interfere with septal development is preferable.

In the transactions of the 1916 meeting of the Oto-Laryngological Section of the American Medical Association there is printed a paper by Dr. Chester C. Cott of Buffalo, entitled "Xanthosis and Other Septal Hemorrhages."

The treatment is somewhat similar to the one just described, and consists of incision and elevation of the mucoperichondrium, followed by the introduction of iodoform gauze, which is snugly packed between the septum and the perichondrium. This is left in position for two days and replaced if bleeding follows its removal.

He reports thirty-eight cases, all of which were ultimately cured.

The introduction of packing between the septum and elevated flap seems to us unnecessary, and the possibility of causing recurrence of the hemorrhage when the packing is removed would seem to be a disadvantage.

Most of the present writer's cases are private patients, and have been seen from time to time during the past three years. The first private case was operated upon December 4, 1913, although several cases had been previously operated upon during the earlier part of 1913, at the Vanderbilt Clinic.

All of the patients have been free from recurrence of hemorrhage except one, a woman aged seventy-five years, whose blood pressure was two hundred and thirty at the time of operation.

The bleeding was checked at the time, but recurred six months later. Reoperation resulted in permanent cure.

The question often arises whether it is advisable to check a moderate degree of bleeding in cases of this kind. The patient insisted upon having relief from the inconvenience of repeated attacks of nose bleed, although probably these were merely physiologic.

One of the series was a case of acute hemophilia, possibly due to pyorrhea alveolaris.

The patient was a stock broker, aged forty-eight years, somewhat alcoholic and otherwise careless of hygiene, although his blood pressure was not excessively high.

Elevation and compression checked the bleeding from the anterior portion of the septum, but two days later blood appeared in the pharynx, and a bleeding point was discovered in the posterior portion of the inferior turbinate. Fresh blood also appeared in the stools, and evidently came from the lower bowel. He was given several injections of human blood serum by Dr. J. E. Welch of this city, and the hemorrhage ceased, although it was deemed advisable to keep the nose packed for about two weeks.

Later on the pyorrhea was treated, and up to the present time, a period of about one year, there has been no recurrence of bleeding.

Several of the writer's colleagues at the Vanderbilt Clinic have successfully used this method of treating septal hemorrhage, and have been able to verify the permanence of the cure in all cases which could be followed up.

SUMMARY.

The object attained by incision and elevation of the mucoperichondrium is free surgical access to the bleeding area, and the rational treatment of a bleeding vessel is compression, causing obliteration of its lumen.

Compresson en masse with a flat bladed forceps reaches all parts of the membrane, and if thoroughly done results in permanent cure.

It is one of the most satisfactory methods of managing cases of septal hemorrhage in children, since the operative treatment

is completed at one sitting, and no prolonged after-treatment is required.

Although the number of cases reported is comparatively small, the evidence adduced by the writer and others is so favorable to this method of operation that it is confidently recommended as a routine procedure in appropriate cases.

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XXII.

SOME INTERESTING LABYRINTH AND EIGHTH NERVE CASES.*

By H. B. Graham, M. D.,

San Francisco.

We have all of us in our examinations of the eighth nerve neuritis cases been struck with the multiplicity in the causes and in the variation in the symptoms. The diseases of all the cranial nerves seem to follow about the same course, exhibiting symptoms and ability to recovery varying according to the function of the nerve, to the quality of the cause of the lesion, and to the intensity of the attack. I have in a former publication pointed out these qualities in respect to the sixth nerve, which have been so ably reviewed by Passow and others, and they have been referred to in the description of the peculiar menieriform polyneuritis of the cerebral nerves described by Frankel Hochwart. For the sixth nerve the causes have been proven by autopsy to be: First, pressure, as from a tumor; second, serous meningitis from middle ear infection; third, the effect of suppurative processes at the apex of the petrous portion of the temporal bone; fourth, toxic, as from lues, lead, arsenic, alcohol and tobacco, or from intestinal toxemia. We can readily see how these same causes might act on any cranial nerve, and especially on the eighth, but in addition to these we have arteriosclerosis, heart lesions, and a nerve exhaustion appearing as prominent factors with the eighth, factors which have not been mentioned in other cranial nerve neuritides. The eighth being so peculiarly mixed in its function, and so intimately connected with other portions of the nervous system, exhibits more pronounced susceptibility to remote influ-

*Read before the Western Section of the American Laryngological, Rhinological and Otological Society, Colorado Springs, Colo., March 17, 1917.

ences and shows a larger variety of easily demonstrable symptoms than do other cranial nerves, and becomes a very important though much neglected factor in the diagnosis of various diseases. This group of cases exhibits a partial or total, temporary or permanent, deafness which has a tendency to recurrence, during which the functional testing may give anything from a middle ear disturbance to that of the typical lesion of the internal ear. Accompanying this may be an apoplectiform disturbance of the vestibular apparatus, as exhibited by dizziness and disturbances of equilibrium, inequalities in the after-turning nystagmus, caloric and other galvanic reactions, and interference with the kinesthetic sense. Either the cochlear or the vestibular symptoms may be uppermost, or one apparatus or the other affected separately, making the lesion as a whole overlooked or referred to other parts of the body, as is often the case when the patient is superficially examined.

To illustrate:

Case 1.—A laborer, thirty years of age, appeared at the Stanford Clinic, complaining of an inability to navigate properly and with a ringing in one ear. He appeared to be drunk, and was examined with that in view, with a result which entirely exonerated the alcohol. His right ear was nonreactive to caloric, and he was deaf in this ear. There was a spontaneous horizontal nystagmus to the opposite side. These symptoms continued with diminishing intensity for a month while the patient was in bed, and when he left the hospital were still present to a marked degree. Three months later he returned and announced that a week following his dismissal from the hospital he had found that his liver was out of order, and had taken . ıe China medicine with prompt secession of all symptoms. While he was in the hospital exhaustive examinations were made of the urine, feces, blood, spinal fluid, and complete physical examination by several internists, and at no time could any assignable cause be given for the neuritis, nor was there anything in his history which would point the way.

Case 2.—F. E., a laborer of thirty-five years, came to me complaining of a dizziness so great that he had been in bed for three days. He gave a history of having had three years previously a headache lasting two years, followed by a paralysis

of the sixth nerve. One month before the paralysis came on the headaches became less, and ceased on recovery from the paralysis, one week later. On examination I found perfect hearing in both ears, no tinnitus; functional tests normal, save a slightly shortened bone conduction; knee jerks were all right, but the patient says they were gone one year before. He has a six-year-old child in whom I found an absence of knee jerks, but who was otherwise normal, as was also his wife. The patient exhibited a constant spontaneous horizontal nystagmus to the right, an absent caloric on the left; turning to the right produced a few jerks, turning to the left gave an extra nystagmus of fifteen seconds to the right. The next day the spontaneous nystagmus was much less, and turning in either direction gave a marked nystagmus to the right; turning to the right, a nystagmus of fifteen seconds, and to the left, a nystagmus of twenty-five seconds. There was a very slight caloric from the right, none from the left ear, so that the right vestibular nerve was now involved with the left. Hearing was perfect. Five days later the spontaneous nystagmus was gone, caloric was slow from either ear, turning always gave a nystagmus to the right of the same value as before. One and two months later the caloric was prompt from the right ear and absent from the left; the turning nystagmus was normal in direction, of small excursion and of a value of twelve to fifteen seconds. There was a negative blood Wassermann, and the urine showed an excessive amount of indican when he first came in. I placed the man on mixed treatment, but do not consider that it was at all established that there was any luetic influence in the case, and the man himself gave a very clear negative history.

Case 3.—An unmarried clerk of thirty years, a young woman under an excessive nervous strain, without a history or signs of lues, who periodically, with six or eight months' intervals, became dizzy for about a week, had to go to bed and exhibited a nystagmus to the left with an exaggeration on movements of the head, absent caloric on the right and perfect hearing.

Case 4.—An active business man of sixty years, with an arteriosclerosis, negative Wassermann, who gave a history of having previously had a slight cerebral hemorrhage and frontal

headaches for twenty-five years, came complaining of unsteadiness in his walking. Functional tests gave Weber unlateralized, Rinne positive, and Schwabach slightly short; watch not heard; forks well heard; monocord at twenty-three. On turning to the right there was little or no nystagmus, but marked dizziness. On turning to the left there was a nystagmus to the right of twenty-two seconds, but the patient was not so dizzy. There was a slow caloric with small subjective signs from the left, and a prompt caloric and marked subjective signs from the right. In other words, there was an incomplete neuritis of the left vestibular nerve, due to an arteriosclerosis; and on testing this left end organ by turning there was a minimum nystagmus, but marked dizziness; and by the caloric test, both a small nystagmus and minimal subjective signs.

I have selected these cases from varied classes and ages. Probably none of them are luetic. No two exhibited similar nystagmus. None of them happen to have symptoms from the side of the cochlea, but these are well known and frequently found, so I need not burden you with them. What rôle interferences in intestinal gland secretion have in the production of these disturbances is not known, but there seems to be evidence pointing to them as the immediate cause in many cases, and the subject is under study at the present time.

I wish now to call your attention to a case exhibiting a peculiar fistula symptom, for which I have no explanation.

Case 5.—A telegrapher of thirty-five years, gave a history of having, seven years before, had a dizziness in blowing his nose, which lasted for four or five minutes, and no further disturbance until about three days ago, when he noticed the same thing. He had a marked coryza when seen; the day before, the telegraph instrument caused a sensation of pain in the right ear. There was a hissing noise in the right ear, both ear drums were slightly injected but transparent. The right drum showed some clouding at the inferior half and an atrophic area in the superior posterior quadrant. The left ear was normal; the right showed slight shrinking for the watch and whisper, seventeen and a half for monochord, fourteen and a half on the left. Weber was to the right; there was no fistula symptom. On politzerizing this patient, a

marked rotary nystagmus occurred in both eyes, at first lasting a couple of minutes to the left, and then changing to the right and lasting another two or three minutes; the patient broke out in a profuse perspiration and became markedly nauseated, this lasting for an hour. The whole phenomenon was repeated three days later. Ten months later the patient heard normally in both ears, monochord at fourteen. There was no disturbance on politzerizing. I present this case without comment for your discussion.

Case 6.—There is another type of case which I wish to call briefly to your attention, and that is an acute temporary disturbance of the cochlear apparatus, due to nerve exhaustion or to acute infection in which there is an acute shrinking of the high tones and a shortened bone conduction, both of which pass off in due time, after a rest or recovery from the infection. I first noticed this in an overworked insurance agent who had had some disturbance in hearing before I saw him. He came to me complaining of an impairment of hearing, and in the course of a week I watched and demonstrated to others the rapid loss of perception for the monochord until he could no longer hear it at all with the rub, and only partially with the bow. This cleared up entirely after a couple of weeks' rest from a very nerve racking occupation.

Case 7.—The next case of this type I saw was an infection with socalled influenza (not a bacterial diagnosis) where there was a shrinking of the perception for the monochord to thirty or less, and a marked shortening of bone conduction, both of which cleared up in a week.

Rapid involvement of the labyrinth in an acute suppurative process is not frequent, but does occur, as illustrated by the following case:

Case 8.—A thirty-four-year-old office employee while in perfect health contracted a fever and became dizzy. On the following day he was told by a physician that he had tonsillitis, although the patient was not aware that there was any disturbance in his throat. On the third day the left ear drum ruptured spontaneously, and the ear continued to suppurate until seen by me, five weeks later. For the first three weeks there was an extreme dizziness and nausea with vomiting, so much so that he thought he was going to fall out of bed. The vomit-

ing ceased after the first week. My examination showed an acute mastoiditis with a nonreactive labyrinth throughout. A Neuman labyrinth operation was done, with no dizziness immediately succeeding the operation. The organism was a capsulated streptococcus. The invasion must have taken place at the very beginning of the infection, but by what route I cannot say. He evidently had not previously had any disturbances of the ear, and no fistula could be demonstrated in the labyrinth capsule at operation. It is problematical as to whether drainage of the antrum on the second day would have saved this labyrinth, but I am inclined to believe that were I to see a case exhibiting these symptoms again, I would strongly advise such an early procedure.

Case 9.—Lastly, I wish to relate a case exhibiting the aural calamities that an individual may be subject to. A man having fallen seven floors, three years and a half ago, sustained a right suppurative ear. Two months later he was sandbagged and was dizzy for four days, so much so that he could not work. This passed off partially, the patient becoming steady by day but not at night. After the sandbagging he was stone deaf in the right (suppurative) ear, as determined by a physician. At the examination it was determined that there was considerable impairment of hearing in the other ear, the high tones being short. The caloric in this good ear was absent, there was no nystagmus on turning in either direction. The patient pointed promptly without error at the object after turning. I take it that the man had a suppurative labyrinthitis in one ear and a concussion affecting the vestibular apparatus in the other. A labyrinth operation on the right was succeeded by no dizziness. The patient exhibited the interesting phenomenon of being unable to orientate himself in space, as when diving in a tank of water.

XXIII.

ACUTE SUPPURATING MASTOIDITIS WITHOUT TYMPANITIS—PERISINUS ABSCESS, PHLEBITIS, STREPTOCOCCEMIA, OPERATION, RECOVERY.

By Charles H. Long, M. D.,

Chicago.

Acute inflammation of the mastoid process, accompanying or following suppurations of the middle ear, is a common occurrence, but is rather rare when we have an insignificant cause and no discharge from the external auditory meatus. The following case, which nearly terminated fatally, very well illustrates such cases, and reminds us of the rapid extension of certain infections without any distinct set of symptoms indicative of any one definite condition.

E. G., female, aged twelve years, student. Entered the Post-Graduate Hospital at two-thirty p. m., May 5, 1916, suffering from chills, fever, vomiting, great prostration and severe pain in the left ear.

About the middle of April she had a severe head cold; later in the month she complained of being cold and chilly, and frequently would be found sitting over the hot air register to keep herself warm. On the afternoon of May 1st, she had to leave school because of a severe pain in the left ear. The family physician, Dr. Robert Black, was summoned. Everything within the knowledge of those present was done to alleviate her suffering, but in spite of all treatment she grew gradually worse; chills, fever, retching, gagging, vomiting, sleeplessness, and delirium. This was the history submitted on her entrance to the hospital.

About four o'clock I found her exceedingly restless, with excruciating pain and tenderness over the left mastoid region. Temperature, 104.2°; pulse, 120; respirations, 36. No meatal

discharge, no tympanitis, membrane red and injected. The nose and throat normal. I freely incised the ear drum, inserted a wick drain and applied external dry heat to the mastoid.

May sixth, after the paracentesis, the pain moved to the opposite side of the head and continued with great severity until I gave her ten grains of aspirin. While she had occasional pain in other parts of her body, none appeared in the head sufficiently severe to demand any special attention. The gauze was removed from the meatus, discolored with only the dried blood from the incision. The drain was not repeated.

The seventh and eighth found her free from head pain, but seemingly more toxemic, sleepless and delirious. The night nurse discovered her walking in the corridor of the hospital. There were great variations of temperature, the maximum being 104.6° at twelve o'clock noon; then she was seized with great pain in the region of the appendix and there was involuntary urination. Blood count: leucocytes, sixteen thousand.

On the ninth, after a conference with Dr. Black and the family, we decided to have Dr. Julius Grinker in consultation. On the tenth we got Dr. Grinker's report—Lateral sinus thrombosis. An X-ray was not taken because of the pain and soreness of the muscles on movement and her great prostration. Indications of meningitis were more pronounced, and there was every reason to believe a fatal termination would ensue unless an effective operation was performed.

At noon on the eleventh the simple mastoid operation was performed; the inner table was found very soft—carionecrotic. The greater portion was removed with the curette, exposing small pools of pus and masses of exudate which completely hid the sinus from view. This was inspected for fistula and examined for tension. The bone was removed in every direction until healthy dura appeared. Now that we had the vein uncovered, from the knee to the tip of the jugular bulb, I proceeded to examine the sinus with a curved applicator, pressing over the vessel to detect a thrombus, but it seemed to be free from coagula. However, to make certain, I made a long incision in the sinus. The blood gushed freely from both ends —no clots appearing, the hemorrhage was controlled by packing. Satisfied with the completeness of the operation, a moist dressing was applied and the patient sent to her room.

May twelfth, while no marked improvement had occurred, she was certainly no worse. The tongue was moist and the meningeal irritation had not increased. The laboratory reported streptococci in the pus.

May thirteenth, there being so little change in her condition, I made a spinal puncture, and found the spinal fluid normal and not under pressure. From then on I was inclined to adopt the "watchful waiting" method of treatment, which in this particular case seems to have met with success. As her blood was freed from the poisonous and septic bacteria, her system became normal and her organs performed their functions in a regular and orderly manner. She was able to leave the hospital June 3d—twenty-three days after the operation, or thirty-four days from the date of leaving school.

The following points in the case are worthy of especial consideration:

First, the mildness of the primary infection.

Second, the transmission of such virulent bacteria from the nose to the mastoid cells without injury to any of the structure during the journey.

Third, the six days of severe pain and tenderness over the mastoid region, then an abrupt cessation.

Fourth, the extensive necrosis of the mastoid found at operation.

Fifth, the fact that no thrombus was found in the sinus.

Sixth, the uselessness of nasal douches and sprays as a prophylaxis of ear infection.

Seventh, the tardiness in operating.

Eighth, the slow convalescence.

A common cold in the head so seldom sets up a mastoiditis without first attacking the middle ear, perforating the drum membrane, and delivering a secretion from the meatus, that I feel we are justified in deferring operative procedures until we have decided upon its necessity and the probability of its success.

It is now quite clear that about May 1st this infection had located itself within the mastoid cells. These cellular walls, being bony, withstood the strain of the violent septic process for six days, when suddenly they weakened and the contents made their way in the direction of least resistance, which was

the dura. The pain and tenderness subsided when the pressure was removed, but the septic and meningeal symptoms continued with increased severity. There was good reason to suspect a sinus thrombosis, with or without extradural abscess, or a meningitis, because we had the classical symptoms of sinus thrombosis—viz., chills and fever with its characteristic marked remitting course, as indicated by the temperature chart; and accompanying these we had the nausea, vomiting, delirium, decided prostration, rapid pulse and respiration, stiffness of the neck, optic neuritis, all of which were very significant of intracranial complications; yet we had not been able to demonstrate the existence of pus in the mastoid by any well defined local manifestations.

In 1907 I witnessed the late Dr. Gruening of New York operate on a young woman manifesting these classical sinus thrombosis symptoms; but the operation, though exposing the sinus very plainly, failed to demonstrate any septic process. That case had made its impression upon my mind, and I was inclined to wait as long as it was safe.

Although Dr. Black gave me entire control of the case, I was glad—for two reasons—of his continued interest: First, and most important, his counsel. Second, to learn the result of the nose and throat cleansing solutions which he had prescribed from the beginning and which I had not opposed. I do not think that many specialists are using them today, because they have learned that these solutions are liable to do more harm than good; and as a prophylactic to ear infections they are worse than useless. They are a relic of the past. As the general practitioner continues to use what he reads, he should be cautioned of their evil effects on the middle ear.

XXIV.

THE PRACTICAL USES OF RECENT WORK ON THE INTERNAL EAR, TO THE GENERAL PRACTITIONER, THE OTOLOGIST, OPHTHALMOLOGIST, SYPHILOLOGIST, NEUROLOGIST AND SURGEON.*

By Isaac H. Jones, A. M., M. D.,

Philadelphia.

Any work in physiology may be regarded as an "interesting study," but from the standpoint of most of us in the practice of medicine, research work of any kind becomes of greatest importance when it proves useful in clinical work. The cry is: "That is all very interesting, but of what use is it? How does it help sick people?" To answer this question is the spirit of this paper. A study of the relation of the ear to the central nervous system not only opens a new field for investigation in physiology, but also furnishes information of diagnostic value in the every-day examination of patients. Although it is essentially and entirely an ear study, and is useful in the analysis of ear conditions, we wish to emphasize that recent investigations have shown it to be of value in medical and surgical diagnosis.

It is now recognized that the internal ear, instead of being one organ, consists of two organs of different functions—the cochlea, which is the organ of hearing, and the static labyrinth, which is the organ of equilibration. It seems very remarkable that whereas the cochlea has long been recognized as the organ of hearing, the static portion of the ear, consisting of the utricle, saccule and three semicircular canals, has been through all the years a "terra incognita." It is only in the past

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few years that the function of the static labyrinth has been carefully studied, preeminently by the Vienna group of otologists, to whom we are indebted for the new methods of testing the internal ear. Robert Bárány received the Nobel prize of 1915 for this work, and he is to be regarded as the pioneer in the clinical application of the study of the ear and the central nervous system.

THE TESTS.

The semicircular canals may be stimulated by revolving a person in a turning chair, or by douching the ears with either cold or hot water, or by applying the galvanic current to the ear. According to our experience, the turning and douching are much more useful than the electric test. The galvanic current stimulates equally all portions of the internal ear, and in fact the eighth nerve itself. As we regard it of greatest importance to analyze the function of the canals separately, we will consider in this paper only the tests by turning and douching. The essential feature in the physiology of these tests is that the endolymph within the particular canal or canals stimulated is set in motion on the one hand mechanically by the turning, and on the other hand thermically by the douching. When an individual is turned to the right, the endolymph catches up to the movement of the body, and when the turning chair is stopped, the endolymph continues to move to the right by its own momentum. The physiology of the caloric test may be illustrated by the "thermosyphon" system of some of the popular automobiles. They have no water pump to force a circulation through the radiator; the engine heats the water, decreasing its specific gravity, and causing it to move upward and forward into the radiator, where it is cooled, drops to the bottom of the radiator and continues downward and backward into the engine, thus completing the circuit. Similarly chilling the outer portion of a semicircular canal increases the density of the endolymph at this point and produces a circulation downward.

Such ear stimulation produces certain definite phenomena in normal individuals—a rhythmic jerking of the eyes, known as nystagmus, and a subjective sensation of turning, which may be termed a systematized vertigo. The eyes are always

drawn in the direction of the endolymph movement, and are then quickly jerked back in the opposite direction by impulses from the cerebrum. The subjective sensation of vertigo is always in a direction opposite to the endolymph movement. According to our conviction, there are only two reactions produced by ear stimulation—vertigo and nystagmus; it is because of this vertigo that the individual falls in a definite direction, and also when he attempts to find with his finger an object he has previously touched, he is unable to find it, but "past-points" to the right or left, above or below, depending on the direction and plane of his sensation of vertigo.

It is evident that if stimulation of the ear causes a movement of the eyes, there must be a definite nerve pathway from the ear to the eye muscles. Also if ear stimulation produces dizziness, it is evident there must be a nerve pathway from the ear to the cerebral cortex. The special object of our work has been the attempt to determine the pathways through the brain from the ear. On the basis of over three hundred and fifty pathologic cases, including a considerable number of operations and autopsies, we have come to certain conclusions as to the course of the fibers from the ear through the brain stem, cerebellum and cerebrum. In brief, our conclusions are as follows: The fibers from the horizontal semicircular canals pass through the eighth nerve to the brain stem, and enter Deiters' nucleus in the medulla oblongata. At this nucleus the fibers divide, going on the one hand to the posterior longitudinal bundle, through which they are connected with the various eye muscle nuclei, to be distributed through the third, fourth and sixth nerves to the eye muscles themselves. It is this pathway that is responsible for the eye movement. The other pathway goes from Deiters' nucleus through the inferior cerebellar peduncle to the cerebellar nuclei of the same side, from which it proceeds through the right superior cerebellar peduncle to the decussation of the two superior cerebellar peduncles in the base of the cerebral crura; from this point there are two pathways to the cerebral cortex of both sides, but the main pathway goes to the cortex of the opposite side. The cortical centers that receive these fibers are postulated by Mills to be in the posterior portion of the second temporal convolutions adjacent to the cortical areas for hearing. It is

this pathway from the horizontal canal to the cerebral cortex, passing through the cerebellum, that is responsible for the production of vertigo on ear stimulation. The fibers from the vertical semicircular canals have a different pathway after entering the brain stem, ascending in the pons to a point above the middle of the pons. At this point the fibers divide, going on the one hand to the posterior longitudinal bundle, to be distributed to the eye muscles; on the other hand the fibers enter the cerebellar nuclei through the middle cerebellar peduncle, from which point their pathway is the same as that of the fibers from the horizontal canal. In a nutshell, by the vestibular apparatus we mean the static portions of the internal ears and the pathways from the ears through the brain stem, cerebellum and cerebrum.

IMPORTANCE OF THESE TESTS IN DIAGNOSIS.

1. The general practitioner is constantly confronted with cases of "vertigo." It is surely not overstating the fact to say that at best he can only guess as to its significance. In the light of these new tests, vertigo should no longer be regarded as a vague and general symptom. We should never speak of gastric vertigo, kidney vertigo, cardiovascular vertigo, idiopathic vertigo, or any other generalization which in its ultimate analysis means nothing. Vertigo is definitely a disturbance of the equilibratory apparatus. These tests give us the means of analyzing this apparatus. All conscious sensations are cerebral; headache, from whatever cause, is a disturbance in the brain. Similarly, vertigo is due to an irritation, impairment or destruction of some portion or portions of the ear or its associated pathways. Therefore, just as we examine the urine in suspected cases of nephritis or diabetes, or as we have a Wassermann test made in suspected cases of syphilis, just so these ear tests enable us to analyze the apparatus responsible for dizziness. Instead of guessing, we then know the reason for the vertigo. We do not hesitate to say that it is very rare that a "dizzy" case remains obscure after these ear tests, and in most instances the diagnosis becomes clear and simple. After these tests are made we are able to say, for example, that the patient is dizzy because of toxic impairment of his labyrinth, a labyrinthitis, an organic lesion in some

part of the vestibular tract such as is produced, for example, by a brain tumor, internal hydrocephalus or multiple sclerosis; or in the presence of normal reactions, we can state with assurance that the vertigo is purely functional.

2. Value to the otologist. It is very surprising how much information can be obtained by these tests in the ordinary everyday study of an ear case. Briefly, from the ear standpoint alone, we have by means of the turning and douching tests, produced nystagmus, vertigo, past-pointing and falling, the clean-cut quantitative objective evidence of the function of each part of the static portion of the labyrinth and eighth nerve. A patient complains of deafness; the question arises, is it a middle ear or an internal ear condition? These hard and fast reactions give positive data of the function of the internal ear; then a positive opinion can be given with less reservation. Deaf and dumb institutions obviously should have a turning chair; in these poor unfortunates the functional tests of hearing are so often unsatisfactory; we never feel after making the hearing tests that we are sure the labyrinth is completely destroyed. It is just here that the new tests are valuable; they either do or do not give reactions. The answer is "Yes" or "No." In routine examination, it is, of course, not necessary to conduct all the tests; merely by turning the patient and obtaining twenty-six seconds of after-turning nystagmus, we at once can conclude that both static labyrinths are normal. Very often, however, the nystagmus will be subnormal, and immediately we have a hint as to the necessity for further study. We have no hesitancy in suggesting that if the otologist in his routine work will make such a simple test, he will be surprised to find that in many instances an entirely new light is thrown upon the condition of his patient.

3. Value to the ophthalmologist. The attention of the eye specialist has not been called to the intimate relation between the ear and the eye. He should surely know that a nystagmus of any type and in any direction can be produced "to order" by ear stimulation. There is an absolute and definite relation between the canal stimulated and the type of nystagmus produced. If we wish to produce a vertical nystagmus upward, we place the patient's head over toward the right shoulder, turn him to the left, and there appears a vertical nystagmus up-

ward. If we wish a rotary nystagmus to the left, we douche the right ear with head upright with cold water, or douche the left ear with hot water, and there is produced a rotary nystagmus to the left. Although space does not permit discussing the reasons for this marvelously accurate ear-to-eye mechanism, yet the above gives some idea of the fixed relationship between the semicircular canals and the eye muscles. The "fistula" test serves as an illustration; if there is caries of bone in the outer wall of the labyrinth producing a fistula into the internal ear, or, for that matter, if the stapes is unduly mobile in the oval window, applying a Politzer bag into the ear and causing pressure or suction, causes the eyes to dance violently. Recently a well known ophthalmologist seeing us do this test expressed his astonishment—"I had no idea there was such close relation between the ear and the eye."

THE PRACTICAL APPLICATION OF THESE TESTS.

(a) In the study of eye muscle palsy. For example, loss of conjugate deviation in various directions should always be studied by means of the ear tests. There is a loss of voluntary control; the patient is unable, when asked, to look in a given direction. If the ear stimulus causes the eyes to move in this direction, it shows without question that there are normal pathways from the ear through the eye muscle nuclei and through the nerves to the eye muscles themselves—therefore, the lesion is supranuclear.

(b) Spontaneous nystagmus may be ocular in origin from corneal scars, macular lesions, or eye strain as in miners, in which case the cause is easily determined by proper ocular examination. But what of those cases in which the lesion lies not so near the surface? It is in such cases that the ear tests are essential. Recently there were two babies, both showing a wild oscillatory nystagmus. Naturally it was the ophthalmologist who was called on to determine its cause; he found nothing wrong in the eyes themselves to account for the nystagmus. The ear tests, however, showed a striking difference between the two cases. On one we reported: "The ear tests suggest an irritation of the vestibuloocular tract, but no organic impairment; the prognosis is good." The child had a perfect recovery. The report on the other patient was:

"There is an organic block of the vestibuloocular tracts of both sides in the brain stem; the prognosis is bad." This patient died. The reason for this report was that the first patient on ear stimulation immediately showed a change of the oscillatory nystagmus into a definite rhythmic pull. The ear stimulation, as it were, seized both eyes and caused them to move rhythmically in the proper direction; evidently, therefore, there was no organic block of the pathways. The other case continued to have the oscillatory nystagmus and was entirely unaffected by ear stimulation, showing a definite organic block of the pathways from both ears to the eyes.

4. The ear and the syphilologist. Syphilis is such a prevalent affection that any method of diagnosis deserves thorough investigation. The disease manifests itself in so many forms that although it is comparatively easy to diagnose in certain cases, it is extremely difficult in others. There is probably no other disease where early diagnosis is so important; if detected early, certain well known remedies can often effect a prompt and permanent cure. Prognosis, in fact, depends largely upon how quickly the treatment is instituted. Discovery of the Wassermann test was justly hailed as an extraordinary aid to the clinician, but unfortunately the Wassermann reaction is not absolute; it is common experience to find cases that are clinically undoubtedly syphilitic, and which also improve under antisyphilitic treatment, but which are repeatedly negative to the Wassermann test. Our investigations to date have led us to believe:

(a) That the new ear tests may prove useful in the early diagnosis of syphilis. Many cases referred to us show a beginning very slight but unmistakable impairment of eighth nerve function only a few weeks after the primary infection; had it not been for the delicacy of the new tests, we would in all probability have not been able to detect this impairment for months.

(b) Ear examinations unquestionably are valuable in detecting a beginning involvement of the central nervous system. It is recognized that the nervous system is attacked in five per cent of those infected; we venture to suggest that probably this percentage is much higher, and that many cases are not recognized because there is no obvious involvement, as in locomotor ataxia, facial paralysis or general paresis. The eighth

nerve is particularly vulnerable to this disease, and analysis of the function of the eighth nerve and the intracranial pathways from the eighth nerve may in certain cases demonstrate a beginning involvement of the nervous system several years before it can be detected by any other method.

5. Value to the neurologist. Naturally all the subjects already mentioned are of interest to the neurologist; the determining of the cause of vertigo, the degree of function of the eighth nerve and labyrinth, the study of eye muscle palsies, the analysis of spontaneous nystagmus, and the early diagnosis of syphilis of the central nervous system. In addition, the neurologist has in these tests a very definite help in:

(a) Differential diagnosis between labyrinth and intracranial lesions. The ear tests are indispensable for such differentiation. In so many instances the symptoms of internal ear disturbances and of cerebellar lesions are identical. To illustrate: A patient was given a neurologic diagnosis of lesion of the right cerebellar hemisphere; ear tests gave additional data to the neurologist and demonstrated conclusively that it was a lesion of the right labyrinth.

(b) Intracranial localization. The accuracy of the ear tests has been very surprising in locating lesions in the cerebellar pontine angle, medulla oblongata, pons, cerebellar peduncles, cerebellum and various portions of the cerebrum, including the parietal lobe, temporal lobe and occipital lobe. Neurologists invariably want an eye examination in intracranial cases. We as otologists are not in a position to state the relative values, but the neurologists and ophthalmologists most acquainted with these ear tests assert that of the two methods of approach, very much more definite information can be had from the ear tests than from the eye examination.

6. Value to the surgeon. Perhaps no phase of surgery is so difficult in diagnosis as intracranial localization. It is a sad commentary on intracranial surgery that in a large majority of cases the surgeon must content himself merely with a palliative decompression. The ear tests have proven of real value in the locating of lesions; but in addition they may prove helpful to the surgeon in preventing unnecessary operations. This may be illustrated by the following two cases: A woman appeared to have a tumor of the right cerebellar hemisphere.

This diagnosis was confirmed by neurologic consultants, and the X-ray report stated that it was a cyst in the right cerebellar hemisphere. The surgeon was about to operate, but hesitated because the ear report said it was a lesion of the right labyrinth, and that the cerebellum appeared intact. The woman had a perfect recovery without operation, has had a baby, and has remained in perfect health ever since—two years. Another woman was operated on for carcinoma of the gall bladder; the findings were negative. Subsequent ear examination made it perfectly simple that all her symptoms—nausea, vomiting, diarrhea and consequent emaciation—had been due entirely to recurring attacks of labyrinthitis. She made an excellent recovery. In the opinion of the surgeons most acquainted with this work, no brain should be opened without giving the patient the benefit of the ear tests.

In conclusion, it must be remembered that the work of the ear specialist along the lines of the relation of the ear to medical and surgical diagnosis is only in its infancy. This paper is intended not so much to call attention to what we have done, as to hint at the future possibilities of this study and to indicate how the cooperation of the otologists with those in other branches of medical work can "help sick people."

XXV.

OBSERVATIONS ON AFTER-TURNING NYSTAGMUS.

By George W. Mackenzie, M. D.,

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I am prompted to present a paper on the subject of after-turning nystagmus for the reason that my investigations, covering a period of eight years (1906 to 1914), have resulted in findings contradictory to those of Bárány, which have been accepted without challenge and as final by the otologic profession. I believe the observations of Bárány on after-turning nystagmus have been faulty because of inaccurate technic, and I trust to prove this contention in the course of the present paper. Furthermore, it is my intention to show that the wide discrepancies in the duration of after-turning nystagmus in normal individuals, claimed by Bárány, do not exist. It is because of these marked discrepancies that certain authorities are inclined to accept the labyrinthine tests with some doubt. It is my belief that a review of my findings, which can be readily corroborated by anyone willing to investigate, will tend to dispel the doubt and permit us to accept the turning test as a valuable one in the diagnosis of labyrinthine conditions.

While my intention is to point out certain errors made by Bárány, there is no intention to reflect upon his otherwise excellent service in the field of otology.

Purkinje, in 1820, was perhaps the first to make observations of vertigo and nystagmus on humans by turning. He, however, did not associate the symptoms with the semicircular canals, but with the brain.

It was left to Flourens, in 1842, to discover the law that each semicircular canal produces eye movements in the plane of the canal, and that when more than one canal was irritated simultaneously, eye movements occurred corresponding to the

resultant of the canals irritated. He conducted experiments upon animals (pigeons and rabbits), sectioning the semicircular canals, from which he concluded that the canals are the seat of an organ which normally had a controlling influence upon body movements. In 1861 the same author experimented upon animals, extirpating the cerebral hemispheres, when he found the semicircular canals reacted after the same manner as before, from which he justly concluded that the reactions were entirely independent of the great brain (cerebrum).

Chevreul, in 1831, made experiments much after the same manner as Flourens; he differed, however, in his conclusions. While Flourens believed the pendulum-like motions and deviations of the head and neck from sectioning of the canals were those of irritation, Chevreul believed them to be manifestations of loss of function from destruction. Since then turning experiments have been made by Crum-Brown, Breuer, Mach, Delage, Kreidl, Kubo and to less extent by others.

It remained for Bárány, however, to conduct the more extensive turning experiments on humans, both normal and pathologic, and to assign to the experiments their place as tests in the diagnosis of labyrinthine conditions. Notwithstanding Bárány's painstaking experiments, he has overlooked certain precautions which have led him into uncertainties which might have been avoided had he observed these precautions.

Bárány, on page 18 of his book, "Physiologie und Pathologie des Bogengang-Apparates beim Menschen," recommends the use of opaque spectacles advised by Hans Abels in timing horizontal after-nystagmus. In the following paragraph he tells why it is advisable to have the subject wear the opaque spectacles, viz., to exclude the physiologic nystagmus that results from the subject fixing his eyes in a side position. In the next paragraph, in referring to rotatory after-nystagmus, he discards the opaque spectacles as unnecessary and uses his fixation apparatus, which consists of a head band to which is fastened a horizontal bar extending forward from the forehead plate about fifteen centimeters. At the distal end of this horizontal bar is attached a vertical bar about four centimeters long; the lower end terminates in a small sphere at which the subject is directed to fix his gaze. He then begins the citation of his findings.

At the bottom of page 18 and the top of page 19 he claims to have examined two hundred normal individuals for horizontal after-turning nystagmus after ten turnings to the right, and found the average duration forty-one seconds, while the after-nystagmus to the left averaged thirty-nine seconds. He also records one individual who showed no after-turning nystagmus after ten turns. The maximum was for nystagmus to the right ninety-eight seconds, to the left one hundred and twenty.

On page 19 he says that we see both extremes—the case with hardly any after-nystagmus and the case with two minutes' duration. Under physiologic conditions there exists great differences in the duration of after-turning nystagmus. He claims that it is not infrequent to find the duration of after-nystagmus in one case to be about three or four times as long as that in another.

In sixty individuals examined repeatedly on the same or on different days, he observed after-nystagmus averaging forty seconds; maximum eighty seconds, minimum twenty-five seconds. He found that only one-fifth of the cases with after-turning nystagmus exceeding forty seconds showed a constancy in the duration of the nystagmus; while the difference ranged from ten to thirty-eight seconds. In other cases with an average duration of after-turning nystagmus of less than forty seconds, the variation ascertained at different examinations ranged from four seconds to twenty-three seconds. He rules out fatigue as a possible explanation, since the examinations were made at times sufficiently far apart to allow for ample rest between.

On page 21, second paragraph, Bárány claims that in a number of cases in which he tried both rapid and slow turnings, he found that after ten rapid turns the nystagmus lasted from eighteen to nineteen seconds, and after slow turns from twenty-six to thirty seconds. He qualifies this statement in a subsequent sentence by saying that in half the cases he found the rapid turns produced a longer after-turning nystagmus than slower turns. Lower down on the same page he claims that with a great number of persons the horizontal nystagmus after twenty, thirty, forty, fifty and sixty turns was the same as after ten turns, adding that "it appears that in the average,

ten turns show the highest worth, while with a greater or less number of turns the amount (duration) diminishes." He claims that the amount (duration) of after-turning nystagmus after twenty turns is about equal to that after five turns. He grants, however, that the nystagmus is more pronounced (intensive) after twenty than after five turns. According to Bárány, after twenty or more turns there appears not infrequently a nach-nach-nystagmus (after-after-nystagmus), which begins a few seconds after the cessation of a very intensive after-nystagmus and lasts sometimes a minute.

Concerning dancers, Bárány claims an interesting finding, namely, that in those who habitually turn to the right, in Austria referred to as rechtstänzer, the duration of the horizontal turning nystagmus to the left (thirty seconds) falls considerably shorter than that to the right (forty-nine seconds). Furthermore, he has never seen the after-nystagmus, in such cases, to the left to last longer than that to the right.

On page 25 Bárány confesses that he is unable to explain why at one time the after-nystagmus to the left exceeds that to the right, while at another time the nystagmus to the right exceeds that to the left. He confesses, furthermore, that he is unable to explain why the nystagmus after twenty turns should be less than after ten turns; and, too, why there should occur an after-after-nystagmus, of one minute's duration, to the opposite side following the after-nystagmus. In his attempts to explain these incongruities he frames up a theory which places the blame upon two vestibular centers in Deiter's nucleus.

Since 1907, when Bárány first presented his findings on after-turning nystagmus, authorities generally have accepted his figures as conclusive, on faith, and incorporated them in their general works on otology as well as in their special works on the labyrinth, including those written by Ruttin and by Braun and Friesner. Others, somewhat more independent, have accepted Bárány's findings, but have ventured their own explanations as substitutes for those of Bárány.

Since Bárány is the one who deserves the credit for having made the most extensive experiments with after-turning nystagmus, and is the most recognized and most quoted authority on the subject, I feel justified in paying him the honor by

directing my controversy towards him, in the hope of convincing him and the otologic profession that his findings are incorrect. I wish to call your attention further to the work of Bárány on after-turning nystagmus by citing from a paper presented by him at the Ninth International Otological Congress,* Boston, August, 1912, and subsequently published in the proceedings under the title of "The Relationship Between the Semicircular Canals and the Eye Muscles, the Central Mechanism in Vestibular Nystagmus." In referring to horizontal after-nystagmus he says: "Stabartz Hofer, at my request, investigated a number of normal cases with no spontaneous nystagmus present, to determine the position in which an exactly horizontal nystagmus would be present in every direction of the visual axis. I controlled his findings. We observed that a pure horizontal nystagmus was never present in a position described by Bruning, but that if turning to the right side is considered, there is always horizontal or rotatory nystagmus to the left when looking to the left." * * * "In another series of experiments I have observed that if with a normal person you produce a strong rotatory nystagmus by turning him with the head ninety degrees forward or backward, you will notice that in every direction of vision the nystagmus will be nearly purely rotatory."

In the same proceedings, in a paper on the "Differential Diagnosis Between Labyrinth Suppuration, Serous Labyrinthitis," etc., page 275, under the heading "Remarks 1," he says: "Spontaneous nystagmus is examined by letting the patient look at the doctor's forefinger, held at a distance of three-fourths of a meter."

In his table for differential diagnosis of the mentioned diseases, in the column under suppuration of the right labyrinth since twenty days, also in that after six months, and under serous labyrinthitis, he mentions that the patient has spontaneous nystagmus to the right as well as to the left side.

I have cited Bárány quite fully, in order that we do him no injustice. I shall now list my table of cases with the findings, after which I shall summarize them and contrast them with Bárány's. Finally, I shall attempt an explanation of the rea-

*The paper was presented but not read, owing to his inability to be present at the Congress.

sons for the disparity that exists between his and my own findings.

I have listed a group of normal cases, a group of cases with middle ear conditions, but with normal internal ears, a group with one-sided internal ear destruction, a series of deafmutes with partial and others with complete destruction of both ears, and a number of others (odd cases) that cannot be classed in any of the above groups. A greater number of cases might have been entered in our list, particularly normals, but the records were found to be too incomplete for the purpose. These occurred in patients complaining of symptoms which vaguely suggested the possibility of internal ear trouble, but upon examination were found to be normal from this standpoint. The results in these cases compared with those in our list. More tests were made for horizontal than for rotatory after-nystagmus, for the reason that the patients tolerated the rotatory less. There were many tests made for rotatory after-turning nystagmus upon charity patients, to whom I offered money for the privilege, but after a few attempts they balked, in spite of the offer, so that complete examination in many cases could not be made. I had to omit them from the list. The results, as far as they went, were in accord with those in the list.

The List of Cases.

Among the twenty-four normal cases, and the eleven cases of middle ear trouble with no spontaneous nystagmus or history of labyrinthine involvement, the longest duration of nystagmus to the right after five turns to the left was nineteen seconds in Case 5. The longest duration of nystagmus to the left after five turns to the right was nineteen seconds in Case 13. The shortest duration of nystagmus to the right after five turns to the left was four seconds in Case 18. The shortest duration of nystagmus to the left after five turns to the right was four seconds in Case 18. The average duration of nystagmus to the right after five turns to the left was a trifle under thirteen seconds, while the average duration of nystagmus to the left after five turns to the right was a trifle over thirteen seconds.

The longest duration of nystagmus to the right after ten turns to the left was twenty-nine seconds in Case 22. The

longest duration of nystagmus to the left after ten turns to the right was thirty-one seconds in Case 2. The shortest duration of nystagmus to the right after ten turns to the left was ten seconds in Case 10. Since this case was that of a whirling dervisher and a left turner, we may omit his case for the present and consider the next shortest, that of fifteen seconds in Case 17. The shortest duration of nystagmus to the left after ten turns to the right was sixteen seconds in Case 17. The average duration of nystagmus to the right after ten turns to the left was a fraction of a second less than twenty-four seconds. The average duration of nystagmus to the left after ten turns to the right was twenty-four seconds. The reduction in after-nystagmus to the right in Case 10 (whirling dervisher) brought the figures for the average the slightest fraction under twenty-four seconds. If this case was excluded the duration would equal about twenty-four seconds.

The longest duration of nystagmus to the right after fifteen turns to the left was thirty-two seconds in Cases 7, 20 and 22. The longest duration of nystagmus to the left after fifteen turns to the right was thirty-two seconds in Case 22. The shortest duration of nystagmus to the right after fifteen turns to the left was eleven seconds in Case 10. Excluding this one we have that of seventeen seconds in Case 11. The shortest duration of nystagmus to the left after fifteen turns to the right was twenty seconds in Cases 10 and 11. The average duration of nystagmus to the right after fifteen turns to the left was a trifle over twenty-six seconds, while the duration of nystagmus to the left after fifteen turns to the right was a trifle over twenty-six and one-half seconds.

The longest duration of nystagmus to the right after twenty turns to the left was thirty-six seconds in Case 22. The longest duration of nystagmus to the left after twenty turns to the right was thirty-six seconds in Case 22. The shortest duration of nystagmus to the right after twenty turns to the left was seventeen seconds in Case 10, next to this it was twenty-three seconds in Case 17. The shortest duration of nystagmus to the left after twenty turns to the right was twenty seconds in Case 15. The average duration of nystagmus to the right after twenty turns to the left was a trifle over twenty-eight

seconds. The average duration of nystagmus to the left after twenty turns to the right was about twenty-eight seconds. In no instance where multiple turns were tried did after-nystagmus to either side after five turns exceed that after ten turns. Nor did the after-nystagmus after ten turns exceed that after fifteen turns, nor did the after-nystagmus after fifteen turns exceed that after twenty. There were cases, however, where the figures after a greater number of turns did not exceed those after a fewer number of turns, but these were exceptional.

The widest discrepancies in the duration of the after-turning nystagmus occurred after five turns—the shortest four seconds, the longest nineteen seconds. The next widest discrepancies in duration occurred after ten turns—the shortest, excluding Case 10, was fifteen seconds, the longest thirty-one seconds. The next occurred after fifteen turns, when the shortest duration was seventeen seconds and the longest thirty-two seconds. The least discrepancy occurred after twenty turns, when the shortest, excluding Case 10, was twenty-three seconds and the longest thirty-six. This would indicate that a twenty turn test is more reliable than a fifteen turn test, that a fifteen turn test is more reliable than a ten turn, and that a ten turn test is more reliable than a five.

Above we have referred only to horizontal nystagmus. Concerning rotatory nystagmus, too few cases have been collected from which to form any conclusion. However, from these few we note an average of twelve seconds to each side after five turns; eighteen seconds after ten turns; excluding Case 10, the average is twenty seconds. We note, too, a greater duration after ten turns than after five, and an increase after fifteen turns over that after ten.

In the list there are ten cases of one-sided labyrinth destruction, eight cases complicating chronic middle ear suppuration and two complicating acute middle ear suppuration. Only two cases, Nos. 45 and 47, were tried with multiple turnings. In Case 45 the after-turning nystagmus after fifteen turns fell below the figures after ten, and this was so on both sides. The other case, 47, showed a consistent increase excepting for the rotatory after-nystagmus to the left, when it was found that the duration after fifteen turns equaled that after ten. All ten

cases were turned at least ten times, either with head erect or bent forward ninety degrees, and some were turned after both methods. Of six who were turned for horizontal nystagmus, the longest duration to the unaffected side was thirty-five seconds in Case 39, the shortest ten seconds in Case 40. The longest duration to the affected side was sixteen seconds in Case 45, the shortest five seconds in Case 40. The average to the unaffected side was twenty-four seconds, to the affected side a trifle under twelve seconds. Eight were turned for rotatory nystagmus. After ten turns the longest duration to the unaffected side was twenty-three seconds in Case 41, while the shortest was ten seconds in Case 40. The longest duration to the affected side was thirteen seconds in Case 37, the shortest was five seconds in Case 40. The average duration to the unaffected side was sixteen seconds, to the affected side eight seconds.

There were four cases of syphilis involving the inner ear or nerve; each differed somewhat from the other, so that we are compelled to consider them separately.

In Case 46 there was partial reduction of hearing in both ears, worse in the left, with Rinné positive on both sides. There was relative reduction to turning and galvanism, particularly on the left side. The case was seen during the stage of improvement which followed antisyphilitic treatment.

In Case 44 there was syphilitic involvement of both sides; on the right the hearing apparatus was more involved than on the left, and the vestibular apparatus about equally on both. The after-turning nystagmus to the two sides was markedly reduced after ten turns—i. e., thirteen seconds to the right and twelve seconds to the left.

In Case 48 we have a typical one-sided destruction (right) of both hearing and vestibular apparatus. After ten turns the horizontal nystagmus to the right was seven seconds, to the left fifteen seconds. The other findings, fourteen and twenty seconds, and those for rotatory nystagmus of thirteen and fourteen seconds, were ascertained late in the case—after the patient had improved somewhat. The findings seven and fifteen seconds were at the height of the affection. This patient subsequently improved considerably with the administration of iodid of potassium internally. I might say, in passing,

that we have found recent cases of syphilitic ear manifestations quite amenable to treatment.

In Case 49 we have a late syphilitic affection of both ears, with marked reduction of hearing and marked reduction of the vestibular function, as shown by the turning test. This patient was turned on several occasions and presented consistently the same findings. After ten turns the horizontal after-nystagmus was twelve seconds to each side. The figures after five turns about equal those after ten turns; after fifteen turns the figures increased slightly; after twenty turns they increased still more than after fifteen turns. In this respect—that is, increasing after fifteen and after twenty turns—she behaved like the normal cases.

Thirty-two deafmutes were examined and are here listed, among which nine were attributed to meningitis. All but one of the meningitis cases were completely deaf on both sides. Case 58 had slight remains of hearing on both sides. All but one case, 54, showed absence of reaction to turning. That is, after ten turns the patients manifested no nystagmus or dizziness. Ten cases of deafmutes were of congenital origin; Cases 61 to 70, inclusive. Of this number three had no reaction from vestibular apparatus; they were Cases 61, 64 and 70. Seven cases showed complete bilateral deafness, and three escaped with slight remains of hearing. Those escaping were Cases 62, 67 and 70. The comparison of these findings shows that the cochlear apparatus is more vulnerable than the vestibular, and in our group may be represented by the proportion of seven to three. In our list of deafmutes we have five cases, from 74 to 78, inclusive, where the cause is assigned to head injuries. One case, 78, shows complete deafness on both sides. Three cases, 74, 76 and 77, have slight remains of hearing, and one case, 75, shows complete deafness on the left side and slight remains on the right. Two cases, 74 and 78, had no reaction to turning. The others showed positive but reduced reaction to both sides. Two cases, 71 and 72, were due to measles. Both were completely deaf on both sides. Case 71 presented no reaction to turning, and Case 72 presented positive reaction to turning, but with reduced figures. One case, 79, was deaf following scarlet fever. The child was completely deaf on both sides, with no reaction to turning. One case, 80,

was completely deaf in both ears following typhoid. This case manifested no reaction to turning. One case, 82, attributed to whooping cough, was completely deaf in the right ear, but had slight remains in the left. The patient presented no reaction to turning. Three cases, 52, 73 and 81, were deaf from unknown causes. All three cases showed slight remains of hearing in both ears. Cases 52 and 73 showed positive but reduced reaction to turning. Case 81 showed no reaction to turning.

Case 83 was an odd one. The patient claims that his deafness came after a cerebral hemorrhage. If he is right in this we must conclude that the deafness was of central origin, the lesion being on the left side. His vestibular function escaped, for he shows a relatively normal after-turning nystagmus to the two sides.

Case 85 deserves special mention. She was a girl, seventeen years of age, who had developed acute mastoiditis following the syringing of the right ear for the removal of wax, but not at my hands. When seen by the writer the patient was suffering from pain, fever, marked tenderness over the ear and mastoid. At the time of operation the prominence of the horizontal semicircular canal showed a fistula. This was anticipated before operation, on account of the already existing horizontal nystagmus to the left becoming decidedly more pronounced by finger pressure in the left ear. It is impossible to include everything in the table, but the end results were remarkable in that she retained function in the vertical canals, so that the case may be classed as one of circumscribed labyrinthitis.

In our list are nine cases of bilateral middle ear suppuration. Cases 84, 93, 94, 95, 96, 97, 101, 106 and 117. Case 94 showed healing on the left side. The hearing was more or less reduced in all nine cases on both sides. Four cases, 95, 97, 106 and 117, showed undeniable reduction of after-nystagmus to the two sides. Case 97 was tried after multiple turns for both horizontal and rotatory nystagmus. Two cases, 84 and 96, showed relatively normal reactions to turnings; the rest showed more or less reduction to one or both sides. Except in Case 84 none showed figures above the average, and this case did not exceed the high marks of the normals. Where multiple

turns were tried, we find again an increase in the duration of the nystagmus with an increase of the number of turns up to twenty. There were occasional differences on the two sides when we compared the horizontal with the rotatory, but this could readily be expected in cases nearing a circumscribed labyrinthitis.

There were four cases of chronic middle ear suppuration. Cases 98, 99, 100 and 113. Case 99 showed marked deafness of the perceiving apparatus type. This, taken with negative reaction to cold water and with reduction of the after-nystagmus particularly toward the affected side, forced me to the conclusion that the inner ear of that side was completely destroyed. The after-nystagmus to the right, after ten turns, was six seconds, and to the unaffected side twelve seconds. This is the characteristic findings of a recent labyrinth suppuration. In contrast with this case is that of Case 113 with nearly normal findings for after-nystagmus in spite of the history of vertigo. The case was one of right-sided chronic middle ear suppuration. Multiple turning tests were made. The figures after fifteen turns were greater than those after ten turns; however, they diminished slightly after twenty turns. Case 100, with chronic middle ear suppuration of the right side and with the history of vertigo, showed spontaneous nystagmus to the right side, more horizontal than rotatory. Also the horizontal nystagmus to the right exceeded the average normal after ten turns to the left, and exceeded considerably the horizontal after-nystagmus to the left. This would suggest a circumscribed irritative process in the right inner ear, probably located in the region of the external semicircular canal. Case 99 represents a still different type of internal ear affection in the right ear secondary to a chronic middle ear suppuration of that ear. The after-nystagmus to the right is diminished slightly. Clinically it would represent a slight or moderate degree of destruction of the semicircular canal apparatus. The after-nystagmus to the right, affected side, after ten turns is eighteen seconds, while that to the unaffected side is twenty-three seconds. In this small group of four cases no two are alike, from the standpoint of the internal ear.

In the list there are four cases, 88, 89, 102 and 110, of acute mastoid abscess. In Case 89, with mastoiditis on the right

side, the after-nystagmus on that side is reduced after ten turns to ten seconds, while it is twenty-four seconds to the unaffected side. The pus was held under considerable tension on this side. There is with it a reduction of function similar to that produced in the cochlear apparatus by pressure with a Gellé bag. The figures approached normal after release of the pressure by operation on the mastoid. This same argument holds true with Case 110, which showed, prior to operation, a horizontal after-nystagmus of fourteen seconds to the right (affected side), and twenty-one seconds to the left (unaffected side). About the same figures were ascertained for the rotatory nystagmus. Both the horizontal and rotatory nystagmus were studied after ten turns. Case 88 approached the normal figures for horizontal after-turning nystagmus. Case 102 was tried after multiple turnings; the figures are relatively low, which at present I am not prepared to explain; however, they follow the rule, in that the figures increased with increased turnings.

Excluding the deafmute cases already considered, there are four cases of basal fractures involving the temporal bone. Cases 50, 87, 104, 107. Case 50 was seen eleven days after the injury, with pronounced mastoiditis and complete deafness on the right side. Vertigo was pronounced as well as a spontaneous nystagmus to the unaffected (left) side. This patient was operated shortly afterward, and at the operation a complete labyrinth operation was performed. Repeated examinations were made, with the result that the patient showed a horizontal after-nystagmus to the right, after ten turns, of eight seconds; to the left of eighteen seconds. After fifteen turns the figures increased, and after twenty they increased still more, following the general rule as ascertained from the normals; so that I am forced to conclude that what is true of two labyrinths is more or less true of one. Case 104 was likewise studied very carefully after a general surgeon had unnecessarily removed the stapes from the left ear when operating for a basal fracture. The patient immediately manifested the symptoms and signs of an acute internal ear destruction, which improved gradually. However, neither the acoustic nor vestibular functions reacted normally. In most respects the case resembled a serous labyrinthitis. This patient

was turned many times and on many occasions. He was used as a demonstration case before classes. Continuous improvement was noted from time to time up to a certain point. The examinations here recorded represent the case about three months after the injury. It will be noted that the duration of the nystagmus is diminished throughout, but particularly to the left (affected) side. It is noticeable, too, that the duration for both horizontal and rotatory nystagmus increases with the number of turns. The patient showed an absence of caloric reaction early, but in time it gradually became positive. Case 87 represents one of basal fracture of one year's standing; reduction of hearing on both sides, particularly on the left. Rotatory spontaneous nystagmus to the right corresponding to destruction of the left inner ear. His after-turning nystagmus to the right is reduced. This patient continues to have vertigo and nystagmus, which suggests that the left inner ear was not completely destroyed at the time of injury, but that there is a continuous or progressive destruction which is not yet complete. This is supported by the entire findings. Case 107 represents a long standing one of basal fracture, with loss of hearing and probable loss of vestibular function. Unfortunately, the caloric test was not made, the patient objecting to further tests and failing to report later.

There was one case, 115, of ocular vertigo relieved by glasses. He presented normal findings and was well balanced to the two sides after ten turns.

There were two cases of otosclerosis, 91 and 92. They both presented the usual findings of marked impairment of hearing on both sides with tinnitus. Case 91 had vertigo. Case 92 had not. There was apparent increase of reaction to the right ear from turning to the left in Case 91, and a pronounced reduction in both ears in Case 92.

There was one case of moderately acute otitis media which improved promptly after a free paracentesis. He was examined several weeks after recovery, so that he might have been, except for the history, classed among the normals. His after-turning nystagmus to the two sides was above the average for normals, but not in excess of the high figures which are rarely found in normals. After fifteen turns his nystagmus was longer than after ten turns; however, the figures diminished somewhat after twenty turns.

I will not occupy the time and space to discuss the details of the other few odd cases; they can be readily referred to in the list.

In all, one hundred and seventeen cases were examined carefully. They include pretty nearly all the varieties of ear diseases to be met with in the average practice. I hope to increase the number of experiments in the future, but I do not see how they will materially affect the general results thus far obtained. The impetus that led me to make and collect these experiences was the fact that I had early found my results with after-turning nystagmus differed from those of the accepted authorities. Had I thought in time to make complete records of all cases, I would have had a much larger amount of material to call upon than is here presented. Since starting the work, what I have lacked in material I have tried to make up for in accuracy of the work.

Let us now compare the findings of Bárány with my own, and note in what particulars they differ:

Barany's Findings in Normals.	My Findings in Normals.
Average duration of horizontal after-nystagmus to the right after ten turns to the left was forty-one seconds.	**Average duration of horizontal after-nystagmus to the right after ten turns to the left was a small fraction of a second less than twenty-four seconds.**
Average duration of horizontal after-nystagmus to the left after ten turns to the right was thirty-nine seconds.	**Average duration of horizontal after-nystagmus to the left after ten turns to the right was twenty-four seconds.**
The maximum duration for horizontal after-nystagmus to the right after ten turns to the left was ninety-eight seconds.	**The maximum duration for horizontal after-nystagmus to the right after ten turns to the left was twenty-nine seconds.**
The maximum duration for horizontal after-nystagmus to the left after ten turns to the right was one hundred and twenty seconds.	**The maximum duration for horizontal after-nystagmus to the left after ten turns to the right was thirty-one seconds.**
Bárány makes mention of seeing one case with no nystagmus after ten turns, and uses this as his minimum for after-turning nystagmus.	**I have observed two cases with apparently no nystagmus, but upon repeating the test and turning the patient at a more rapid speed and examining carefully, I was able to note and time the nystagmus, when it was found to be of relatively short excursions and lasted the usual length of time.**

In sixty individuals examined on different days, he observed horizontal after-nystagmus averaging forty seconds. Maximum, eighty seconds; minimum, twenty-five seconds. He found only one-fifth of the cases showing consistently the same findings, which is the negative way of putting it that four-fifths showed variable findings.	I have examined a fewer number of cases after such methods, and have found the average, after ten turns for horizontal after-nystagmus, to equal about twenty-four seconds. I have never found anything like the variations that Bárány has. The greatest variations were but a few seconds, and these could readily have been explained on the ground of faulty observations.
In a number of cases where he had tried both rapid and slow turnings, he has found that after ten rapid turns the nystagmus lasted from eighteen to nineteen seconds. While after slow turns, from twenty-six to thirty seconds. In one-half the cases he found the rapid turns produced a longer after-nystagmus than slower turns.	I have tried patients with both rapid and slow turnings, with the result that the duration is apparently less after slow than after rapid turns, and consistently so. However, most careful tests will show them to be about equal in duration. The difference is more apparent than real, for the reason that after slow turning the nystagmus is less intensive than after rapid turning, hence less noticeable and more difficult to determine the moment of cessation.
He claims that with a great number of persons the horizontal nystagmus after twenty, thirty, forty, fifty and sixty turns was the same as after ten turns; adding that it appears that in the average ten turns show the höchsten Werte (highest worth, value or rate), while with a greater or less number of turns the amount diminishes.	I have not examined many persons after as many as thirty, forty, fifty and sixty turns, for the reason that they are hard to get. So I had to be the subject myself many times. I am, therefore, not in the position to match Bárány in material. I have not found the after-turning nystagmus after ten turns to be of the höchsten Werte in any sense in which the expression may be interpreted. For instance, I have found the nystagmus to increase consistently in duration between ten and twenty turns. Furthermore, I find less variations after twenty than after ten turns, showing, therefore, that the results are more reliable after twenty turns than after ten turns.
He claims that the amount of after-turning nystagmus after twenty turns is about equal to that after five turns.	I have never in my experience found this to be true in a single case.

He claims that after twenty or more turns there appears, not infrequently, a nach-nach-nystagmus (after-after-nystagmus) in the direction opposite to the after-nystagmus, lasting sometimes a minute.

Concerning dancers, he claims that with those who habitually turn to the right the duration of the horizontal turning nystagmus to the left falls considerably shorter than that to the right.

I have never seen an after-after-nystagmus in many hundred examinations, including those in my list. I do not believe it exists, and will not believe it until it is shown to me.

I was fortunate enough to obtain a whirling dervisher, with the stage name of Hash Hash, who was a left turner. I examined him on many occasions, and found that his duration of nystagmus was consistently the same on several occasions. The figures were reduced for both horizontal and rotatory nystagmus to both sides, and more so to the right. This one case supports the claims of Bárány. (Case 10 in the list.)

Concerning the theory that Bárány has to offer in explanation of his variable findings, I have only to say that I cannot accept it. I do not see how his findings can possibly be right without my findings, which have been carefully undertaken, supporting his; and if his findings are wrong, they being his premise, then must his conclusions be false. It does not seem likely that nature should provide us with definite centers to carry on such pranks in physiologic behavior as are accredited by Bárány, and that my findings, which are fairly consistent, should be false.

Bárány in his efforts to find a relationship between the semicircular canals and the eye muscles, neglects a certain precaution that he himself mentions when he advises the use of opaque glasses in studying horizontal after-turning nystagmus.

Bárány in his efforts to find "The Relationship Between the Semicircular Canals and the Eye Muscles" makes observations of horizontal after-turning nystagmus in the various positions of the visual axis—i. e., by having the patient direct his gaze in various positions. He also makes observations of nystagmus "by letting the patient look at the forefinger held at a distance of three-quarters of a meter"; all of which is in direct contradiction to the precautions laid down in his original monograph, where he advises the use of the opaque spectacles of Abels.

Together with many other Americans, it was my good fortune to have taken Bárány's course on the labyrinth, prior to and shortly after the publication of his work. At that time the opaque spectacles were not used on all occasions in timing after-turning nystagmus. Many tests were made exactly after the method mentioned by him in the proceedings of the Ninth International Otological Congress—that is, by having the patient look at his forefinger held three-quarters of a meter from the patient, both in front of and to the sides of the patient. He will never be able to determine the relationship between the eye muscles and semicircular canals after this manner, for the reason that as soon as he permits the patient to direct his eyes away from the middle line to one or the other side, no longer can there be a purely semicircular canal reflex, but this reflex plus tension on one or more of the extraocular muscles. A pure semicircular canal reflex nystagmus is manifested only when the patient's eye muscles are in a state of complete relaxation, if such a thing is possible. This condition is most nearly attained when the patient looks straight ahead at some distant object, and not at the finger held three-quarters of a meter from the patient.

What makes horizontal nystagmus to the left more intensive when looking to the left and less intensive when looking to the right? Surely, it cannot be that there is an actual increase or diminution of the intensity of the stimulus originating in the semicircular canals. We must look for a simpler explanation. The answer is that when looking to the left there is the stimulus in the canals which calls forth the reflex plus something else, and that when the patient looks to the right there is the stimulus in the canals which calls for the reflex minus something else. This plus and minus element is found in that normal physiologic nystagmus, or the tendency to it, to the left when looking to the left, and to the right when looking to the right; upward when looking upward, etc. It is well known that the average individual will show a horizontal nystagmus to the left when looking intently to the left, etc. In some cases, instead of a horizontal nystagmus, it may become rotatory in the extreme lateral positions of the eyes. The tendency toward rotatory nystagmus becomes greater the more the eyes are directed away from the horizontal position. I may go

a step farther to prove that this physiologic nystagmus, or the tendency to it, plays an important rôle in influencing the character and intensity of the socalled vestibular nystagmus. For instance, I have repeatedly observed that a relatively intensive horizontal after-nystagmus to the left may be completely annulled by having the patient look strongly to the right. By having the patient look still stronger to the right, the physiologic element overbalances the vestibular nystagmus to the left, when he exhibits a purely rotatory nystagmus to the right. If you have not consumed too much time in your experiment thus far, you may be able to elicit a return of horizontal nystagmus to the right by having the patient look straight ahead. The character and direction of the vestibular nystagmus may be further influenced in the side positions in cases showing muscle imbalance, particularly when that imbalance is due to paretic conditions of one or more of the eye muscles. It is well known among oculists that in cases with eye muscle paresis the patient will manifest a rhythmic nystagmus more pronounced when looking in the direction corresponding to the action of the weak eye muscle than when looking in the opposite direction.

I cannot help but attribute Bárány's irregular findings, as compared to my own, as due to his technic, which I believe is faulty, rather than to actual irregularities in the behavior of the reflexes.

In his "Table for Differential Diagnosis of Mentioned Diseases," page 276, Proceedings of Ninth International Otological Congress, he indicates with arrows the direction of spontaneous nystagmus in the column, suppuration of the right labyrinth since twenty-one days and elsewhere, that the patient has spontaneous nystagmus to the right as well as to the left. A nystagmus to the right in such a condition is impossible unless the patient looks to the right. This nystagmus to the right, when looking to the right, is not in the least way connected with the patient's labyrinthine condition. It is the same physiologic nystagmus that a normal person may manifest. If we have the patient look straight ahead the nystagmus to the right would not be present. That Bárány has included it among his other findings in labyrinth suppuration only supports my previous contention, that he lays more stress upon the side positions than the straight forward position of the

visual axis. He might answer by saying that he compares the intensity of the nystagmus to the two sides, and to aid him in accuracy at obtaining the same angle deviation, he uses his fixation apparatus. My reply to this would be that to ascertain the exact angle to the two sides takes some time, and during the interim one must carry in his mind the exact intensity of the nystagmus to the side previously examined, in order to make comparison; and comparisons that depend on memory ever so short cannot be as exact as those made simultaneously. Another objection is that the instrument must be tried carefully beforehand to see that zero corresponds exactly with the straight ahead position of the eyeballs. Furthermore, the proximithy of the instrument to the eyes causes a considerable degree of convergence (action of the interni muscles), which must of necessity interfere with the free action of the vestibular reflex. Finally, before the findings in the lateral positions of the eyes can be accepted in any particular case, a careful examination of the eye muscles must be made to eliminate possible heterophorias or paresis.

In his table there is no mention made of his having noted the spontaneous nystagmus that occurs when looking straight ahead; while with me no other nystagmus is of value from the standpoint of the semicircular canals. A nystagmus which is elicited by turning movements of the eyes belongs to the eye muscles. If the intensity of the nystagmus balances in opposite directions, I accept it as physiologic; when they do not, I accept it as pathologic, and the problem then resolves itself down to the isolation of the particular muscle or muscles involved.

I might add more arguments to my case, but they would be only corroborative of what has already been said. Accordingly, I will rest my case.

Case 1.—W. L., male, 28 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 11½ seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 21 seconds; after ten turns to the right, with

head erect, horizontal nystagmus to the left 22 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to right 25 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 26½ seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 2.—W. H. B., male, 43 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 29 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 3.—C. P. W., male, 42 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 28 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 31 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 4.—A. H. C. R., male, 45 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 5.—H. A. L., male, 43 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 25½ seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 29 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 20 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 20 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 6.—J. L., male, 13 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 32 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 32 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 7.—G. W. M., male, 43 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 16 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 17 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 8.—A. V. M., female, 45 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 14½ seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 9.—A. K. M., male. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 19 seconds; after turning five times to the right, with head erect, horizontal nystagmus to the left 15 seconds. After turning ten times to the left, with head erect, horizontal nystagmus to the right 21 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 21 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 22 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 10.—H. H., male, 45 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, no nystagmus to the right; after five turns to the right, with head erect, horizontal nystagmus to the left 10 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 18 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 11 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 21 seconds. After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 10 seconds; after five turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 12 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 11 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 13 seconds. After fifteen turns to the left, with head inclined forward about ninety degrees, rotatory nystagmus to the right 12 seconds; after fifteen turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus

to the left 17 seconds. After twenty turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 13 seconds; after twenty turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 20 seconds. Galvanic reaction: Right ear, cathode 6 m.a., rotatory nystagmus to the right; anode 4½ m.a., rotatory nystagmus to the left. Left ear, cathode 4½ m.a., rotatory nystagmus to the left; anode 5 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Dervisher dancer, left turner, generally turns 118 times in a minute, has turned 150 times in a minute; fourteen years ago turned five hours and ten minutes without stopping. The nystagmus is more apparent to the left, making longer and quicker movements.

Case 11.—G. C. W., male, 26 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After turning ten times to the left, with head erect, horizontal nystagmus to the right 17 seconds; after turning ten times to the right, with head erect, horizontal nystagmus to the left 18 seconds. After turning fifteen times to the left, with head erect, horizontal nystagmus to the right 17 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 12.—G. H., male, 24 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 10 seconds. Aften ten turns to the left, with head erect, horizontal nystagmus to the right 18 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 18 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 28 seconds; after twenty turns to the right,

with head erect, horizontal nystagmus to the left 30 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 13.—E. La B., female, 21 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 16 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 27 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 14.—W. W. R., male, 32 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 18 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 27 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 28 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 15.—M. B., female, 16 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmas: After five turns to the left, with head erect, horizontal nystagmus to the right 6 seconds; after five turns to the right,

with head erect, horizontal nystagmus to the left 6 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 28 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 10 seconds; after five turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 9 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 18 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 18 seconds. After twenty turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 21 seconds; after twenty turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 15 seconds. Galvanic reaction: Right ear, cathode 7½ m.a., rotatory nystagmus to the right; anode 4½ m.a., rotatory nystagmus to the left. Left ear, cathode 4¾ m.a., rotatory nystagmus to the left; anode 7½ m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Normal case.

Case 16.—Miss B., female, 20 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after five turns to the right, with head erect, horizontal nystgamus to the left 15 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 27 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. On another occasion: After five turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 16 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 19 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 21 seconds. After twenty turns to the left,

with head erect, horizontal nystagmus to the righ 28 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 2 m.a., rotatory nystagmus to the left. Left ear, cathode 2 m.a., rotatory nystagmus to the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Normal case.

Case 17.—L. P., male, 24 years of age. No vertigo, tinnitus in both ears, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings: Right ear, membrane opaque, slightly retracted, chalk deposit posterior half; left ear, same. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 16 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. Galvanic test not taken. Caloric test not taken. Remarks: Came because of tonsillar condition and repeated attacks of acute secretory catarrh, and examination made during one of these attacks.

Case 18.—F. C., male, 23 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 4 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 4 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 21 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 29 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 32 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 29 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 4 m.a., rotatory nystagmus to the left. Left ear, cathode 4 m.a., rotatory nystagmus to the left; anode 4 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Right-sided Bell's palsy.

Case 19.—M. G., female, 18 years of age. Vertigo present off and on for several years, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 12 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 13 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. Galvanic reaction: Right ear, cathode 7 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 4 m.a., rotatory nystagmus to the left; anode 6½ m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Eye muscles half a degree of exophoria and half a degree of right hypophoria.

Case 20.—F. L., male, 40 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 13 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 13 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 32 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 31 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 35 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 31 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 21.—T. N. H., male, 20 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 27 seconds.

After twenty turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. Galvanic test not taken. Caloric test not taken. Remarks: Normal case.

Case 22.—L. P. B., male, 43 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 29 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 28 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 32 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 32 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 36 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 36 seconds. Galvanic test not taken. Caloric test not taken. Remarks: Normal case.

Case 23.—D. D. C., female, 26 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 27 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 29 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 31 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 34 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 33 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 24.—J. McC., male, 23 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 10 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. After

fifteen turns to the left, with head erect, horizontal nystagmus to the right 27 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Normal case.

Case 25.—M. P., female, 16 years of age. Vertigo at times, tinnitus, buzzing sound in left ear, no spontaneous nystagmus. Hearing tests, impairment both ears, more in the left. Weber to the left. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, membrane slightly retracted and opaque. Left ear, moderate profuse brown offensive discharge. Large perforation anterior inferior quadrant. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 28 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: One-sided middle ear suppuration.

Case 26.—W. B., male, 23 years of age. Vertigo had been present, but not now. No tinnitus, no spontaneous nystagmus. Hearing moderately reduced. Worse in right ear. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, perforation with discharge. Left ear, dry perforation. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 11 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 11 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. Gal-

vanic reaction not taken. Caloric reaction not taken. Remarks: Bilateral chronic middle ear suppuration.

Case 27.—W. S. B., male, 35 years of age. Vertigo present, no tinnitus, no spontaneous nystagmus. Hearing tests, normal findings. Otoscopic findings normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 27 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Case proved to be one of ocular vertigo, which was corrected by lenses.

Case 28.—E. K., female, 12 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, fair both sides. Weber indifferent. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. C_1 not heard on the right and shortened on the left. Otoscopic findings: Right ear, discharge. Left ear, discharge very pronounced, canal narrowed. After-turning nystagmus: After five turns to the left, with head erect, no nystagmus to the right; after five turns to the right, with head erect, no nystagmus to the left. After ten turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after ten turns to the right with head erect, horizontal nystagmus to the left 14 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 18 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 18 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 21 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. At another occasion: After ten turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 16 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Bilateral chronic middle ear suppuration.

Case 29.—C. W., male, 25 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, hearing reduced both sides. Weber to the right. Schwabach lengthened on both sides. Rinne negative on both sides. C_1 shortened on both sides, C_4 normal on both sides. Otoscopic findings: Right ear, membrane intact, secretion showing behind the lower half of the membrane with a niveau line indicating

the level of the secretion. Left ear, membrane red, bulging slightly, perforation in the anterior inferior quadrant. Secretion somewhat thin, moderate amount. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 12 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 29 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 28 seconds. Galvanic test not taken. Caloric test not taken. Remarks: More physiologic nystagmus to the right when looking to the right than to the left when looking to the left. Left acute middle ear suppuration, right secretory catarrh.

Case 30.—R. W., female, 8 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing fair on both sides. Weber to the right. Schwabach very slightly lengthened on the right and on the left. Rinne neutral, neither positive nor negative on both sides. Otoscopic findings: Right ear, normal. Left ear, attic perforation. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. Galvanic reaction not taken. Caloric reaction not taken.

Case 31.—R. W., male, 9 years of age. Vertigo, light attacks on rare occasions, no tinnitus, no spontaneous nystagmus. Hearing tests, very marked reduction both sides. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. C_1 shortened on the right and on the left. C_4 shortened on the right and on the left. Otoscopic findings: Right ear, slight secretion, hammer handle visible, also long process of the incus. Polyps on inner wall. Left ear, profuse, thick, yellow, offensive discharge. Remnants of membrane in front, hammer

handle remains. Granulations in tympanic cavity. Deep opening in niche of the oval window. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 19 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 20 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. Galvanic reaction not taken. Caloric reaction, cold water both sides positive. Remarks: Bilateral chronic middle ear suppuration left side, tuberculous mastoiditis.

Case 32.—C. B. C., male, 27 years of age. No vertigo, occasional ringing noises, no spontaneous nystagmus. Hearing tests, moderate impairment, worse on left side. Weber to the left. Schwabach shortened on the right and on the left. Rinne positive on both sides. Otoscopic findings: Right ear, membrane intact, hammer handle visible. Left ear, total destruction of membrane. Newformed adherent to the inner wall. Head of stapes seen and stapedius muscle. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 13 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. Galvanic reaction not taken. Caloric reaction not taken.

Case 33.—L. S., male, 35 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, moderate reduction on the right, normal on the left. Weber to the left. Schwabach shortened on the right. Rinne negative on the right and on the left. C_4 shortened on the right. Otoscopic findings: Right ear, membrane slightly opaque, otherwise normal. Left ear, the same. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 20 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 23 seconds. Galvanic reaction: Right ear, cathode 6 m.a., rotatory nystagmus to the

right; anode 7 m.a., rotatory nystagmus to the left. Left ear, cathode 7 m.a., rotatory nystagmus to the left; anode 6 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Right-sided seventh nerve palsy; involvement of the eighth nerve, especially the cochlear branch. Developed six months before examination.

Case 34.—S. W., male, 22 years of age. Vertigo one attack; no tinnitus, no spontaneous nystagmus. Hearing tests, reduction in both ears, pronounced on the right, slight on the left. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, slight amount of creamy discharge. Granulations in attic antrum region. Left ear, membrane opaque, normal position, small scar in the anterior superior quadrant. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 10 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 30 seconds. After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 15 seconds; after five turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 14 seconds. Galvanic reaction not taken. Caloric reaction positive. Remarks: Seen in consultation with Dr. Dowling because of continued discharge after radical operation on right side.

Case 35.—W. R., female, 51 years of age. Vertigo, dizziness worse when lying down; tinnitus like roaring of the sea, no spontaneous nystagmus. Hearing tests, marked reduction on the left, normal on the right. Weber to the right. Schwabach slightly shortened on the left. Rinne positive on the left. Otoscopic findings: Right ear, membrane slightly opaque, otherwise normal. Left ear, the same. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 21 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After ten turns to the left, with head inclined for-

ward about 90 degrees, rotatory nystagmus to the right 23 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 19 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Meningitis five months previous.

Case 36.—J. E. O., female, 35 years of age. Vertigo, dizziness slight and of short duration; no tinnitus, no spontaneous nystagmus. Hearing tests: Left ear, reduced. Right ear, slightly reduced. Weber to the left. Schwabach shortened on the right and on the left. Rinne positive on the right, and neutral, neither positive nor negative, on the left. Otoscopic findings: Right ear, membrane normal color but slightly retracted. Left ear, bulging and red membrane, no perforation. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 22 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Subsequent operation for mastoiditis left side.

Case 37.—F. G., male, 24 years of age. Has vertigo, no tinnitus; spontaneous nystagmus rotatory to the right looking straight ahead. Hearing tests, complete deafness on the left. Weber to the right. Schwabach normal on the right and shortened on the left. Rinne positive on the right and negative on the left. C_1 not heard on the left and C_4 not heard on the left. Otoscopic findings: Right ear, membrane slightly cloudy, otherwise normal. Left ear, membrane totally destroyed. Polyps from attic antrum region. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 15 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 7 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 7 m.a., no reaction. Left ear, cathode 7 m.a., no reaction; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction, cold water to the left ear negative. Remarks: One-sided labyrinth destruction. Chronic middle ear suppuration left side, cholesteatoma, fistula of the external semicircular canal, meningitis. Radical and labyrinth operation. Cured.

Case 38.—L. H., male, 33 years of age. Has vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, deaf in the right ear by all tests. Otoscopic findings: Right ear, destruction of right membrane with offensive discharge. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 11 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 13 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 18 seconds. Galvanic reaction: Right ear, cathode 7 m.a., rotatory nystagmus to the right; anode 3 m.a., rotatory nystagmus to the left. Left ear, cathode 4 m.a., rotatory nystagmus to the left; anode 7 m.a., rotatory nystagmus to the right. Caloric reaction, cold water in right ear negative. Remarks: Chronic middle ear suppuration right side, with cholesteatoma, fistula of the external semicircular canal and labyrinth suppuration.

Case 39.—F. K., male, 34 years of age. Has vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, deaf in the left ear by all tests. Otoscopic findings, left membrane destroyed, offensive discharge. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 20 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 7 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 8 m.a., no reaction. Left ear, cathode 8 m.a., no reaction; anode 8 m.a., no reaction. Caloric reaction, cold water to the left ear negative. Remarks: Chronic middle ear suppuration on the left side with labyrinth sequestration. Left-sided facial paralysis.

Case 40.—R. W., male, 27 years of age. Has vertigo, no tinnitus, spontaneous nystagmus, rotatory to the right when looking straight ahead. Hearing tests, deaf on left side by all tests. Otoscopic findings: Destruction of the left membrane with discharge. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 35 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory

nystagmus to the right 19 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 11 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 7 m.a., no reaction. Left ear, cathode 7 m.a., no reaction; anode 4 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Chronic middle ear suppuration left side with cholesteatoma and destruction of the labyrinth.

Case 41.—G. G., male, 37 years of age. Has vertigo with vomiting, no tinnitus, no spontaneous nystagmus. Hearing tests, deafness on the right side. Otoscopic findings, right membrane destroyed with secretion. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 5 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 10 seconds. Galvanic reaction: Right ear, cathode 12 m.a., rotatory nystagmus to the right; anode 6 m.a., rotatory nystagmus to the left. Left ear, cathode 6 m.a., rotatory nystagmus to the left; anode 12 m.a., rotatory nystagmus to the right. Caloric reaction, cold water to the right ear negative. Remarks: Chronic middle ear suppuration right side with destruction of the labyrinth and facial paralysis.

Case 42.—W. P., male, 34 years of age. Has vertigo with vomiting, no tinnitus, spontaneous nystagmus, slight rotatory nystagmus to the left when looking straight ahead. Hearing tests, deaf in the right ear by all tests. Otoscopic findings, destruction of the right membrane. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 6 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 23 seconds. Galvanic reaction: Right ear, cathode 16 m.a., no reaction; anode 6 m.a., rotatory nystagmus to the left. Left ear, cathode 4 m.a., rotatory nystagmus to the left; anode 14 m.a., no nystagmus. Caloric reaction, cold water to the right ear negative. Remarks: Right chronic middle ear suppuration with labyrinth destruction. Radical operation performed together with labyrinthine drainage.

Case 43.—A. K., female, 40 years of age. Has intense vertigo, no tinnitus, spontaneous nystagmus, rotatory nystagmus to the right when looking straight ahead. Hearing tests, com-

plete deafness in left ear. Otoscopic findings, left membrane totally destroyed, offensive discharge. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 30 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 14 seconds. Galvanic reaction: Right ear, cathode 2 m.a., rotatory nystagmus to the right; anode 12 m.a., rotatory nystagmus to the left. Left ear, cathode 12 m.a., rotatory nystagmus to the left; anode 7 m.a., rotatory nystagmus to the right. Caloric reaction, cold water to the left ear negative. Remarks: Chronic middle ear suppuration left side with labyrinth suppuration. Meningitis. Left facial palsy. Radical and labyrinth operations.

Case 44.—E. K., female, 46 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, deaf in the right ear by all tests. Otoscopic findings, total destruction of the right membrane with offensive discharge. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 7 seconds; after ten turns to the right, with head inclined forward about 90 degrees; rotatory nystagmus to the left 14 seconds. Galvanic reaction: Right ear, cathode 6 m.a., rotatory nystagmus to the right; anode 4 m.a., rotatory nystagmus to the left. Left ear, cathode 3 m.a., rotatory nystagmus to the left; anode 7 m.a., rotatory nystagmus to the right. Caloric reaction, cold water to the right ear negative. Remarks: Chronic middle ear suppuration, right side, with cholesteatoma and destruction of the labyrinth. Carcinoma.

Case 45.—W. L. B., male, 31 years of age. Vertigo marked. One spontaneous attack in office associated with rotatory nystagmus to the left, tinnitus in right ear, spontaneous nystagmus present off and on, variable in intensity, always to the left. Hearing tests, right markedly diminished. Weber to the left. Schwabach shortened on the right, normal on the left. Rinne negative on the right and positive on the left. Otoscopic findings: Right ear, membrane slightly cloudy and retracted. Left ear, same, but less pronounced. After-turning nystagmus: After ten turns to the left, with head erect; horizontal nystagmus to the right 13 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Syphilitic lesion both sides, more pronounced right.

Case 46.—S. A., female, 60 years of age. Has vertigo, has tinnitus, worse in left ear, no spontaneous nystagmus. Hearing tests, fair right ear, deaf in left ear. Otoscopic findings: Right ear, slightly opaque. Left ear, membrane slightly cloudy and retracted. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 16 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 14 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Labyrinth suppuration secondary to acute middle ear suppuration. Seen in consultation with aurist, who treated the case conservatively. Examination was made three months after onset of the trouble. A few weeks after this patient was able to be around.

Case 47.—D. L., male, 38 years of age. Formerly severe attacks of vertigo, but at present only occasional slight attacks; tinnitus, buzzing sound, worse at night, no spontaneous nystagmus. Hearing tests, reduction in both ears, more in the left. Weber to the right. Schwabach shortened on the right and on the left. Rinne positive on the right and on the left. Otoscopic findings: Right ear, membrane slightly cloudy, normal mobility. Left ear, same. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 22 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 21 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 7 m.a., rotatory nystagmus to the left. Left ear, cathode 7 m.a., rotatory nystagmus to the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Sequela of eighth nerve neuritis of questionable origin, possibly syphilis. Wassermann negative; the patient gave history of infection (chancroid with bubo).

Case 48.—R. McC., female, 39 years of age. Vertigo pronounced with vomiting, has tinnitus, spontaneous nystagmus

rotatory to the right when looking straight ahead. Hearing tests, complete deafness left side by all tests. Otoscopic findings: Right ear, normal. Left ear, membrane intact, dull and opaque. Anatomic details impossible. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 9 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 4 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 5 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 10 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 11 seconds. After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 1 second; after five turns to the right, with head inclined forward about 90 degrees, no rotatory nystagmus to the left. After ten turns to the left, with head inclined forward about 90 degres, rotatory nystagmus to the right 8 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 6 seconds. After fifteen turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 10 seconds; after fifteen turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 6 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 10 m.a., cessation of nystagmus. Left ear, cathode 10 m.a., no reaction on the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction negative. Remarks: Rapid labyrinth destruction of the left side following a few days after the onset of acute middle ear suppuration.

Case 49.—G. L., male, 52 years of age. Vertigo pronounced at onset, but none at present; tinnitus came on suddenly eight weeks ago, slight at present time; spontaneous nystagmus rotatory to the left. Hearing tests, complete deafness in right side, left side normal. Otoscopic findings negative. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 7 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After ten turns to the left, with head

erect, horizontal nystagmus to the right 14 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 20 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 13 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 14 seconds. Galvanic reaction not taken. Caloric reaction present both sides, but diminished to the right. Remarks: Gave history of syphilitic infection many years ago. At time of test, blood pressure 200.

Case 50.—W. A. S., female, 47 years of age. Has vertigo, tinnitus pronounced, no spontaneous nystagmus. Hearing tests, marked reduction in both ears. Weber indifferent. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: A slight opacity of membrane, but no retraction both ears. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 12 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 12 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 14 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 4 m.a., rotatory nystagmus to the left. Left ear, cathode 4 m.a., rotatory nystagmus to the left; anode 4 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Late lues. Evidences of old interstitial keratitis.

Case 51.—G. D., male, 25 years of age. Vertigo pronounced, no tinnitus; spontaneous nystagmus, rotatory to the left looking straight ahead. Hearing tests, complete deafness on the right side. Otoscopic findings: Right ear, external canal swollen, pulsating discharge. Anatomic details not seen. Swelling over mastoid. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 8 seconds; after ten turns to the right, with head erect,

horizontal nystagmus to the left 18 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 9 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 11 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 28 seconds. Galvanic reaction not taken. Caloric reaction negative on the right side. Remarks: Fractured skull involving base. Seventh nerve paralysis complete. At time of operation fractured fragment of temporal bone involving inner ear was removed.

Case 52.—F. E. H., female, 6 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, deaf in both ears by all tests. Otoscopic findings: Right ear, membrane opaque and retracted; no perforation, no scars. Left ear, same. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. After twenty turns to the left, with head erect, and after twenty turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Ophthalmoscopic examination showed excessive connective tissue on disc, which, together with the history and findings, suggested meningitis at the age of three years.

Case 53.—J. M. K., male, 54 years of age. Vertigo pronounced off and on, tinnitus present, no spontaneous nystagmus. Hearing tests, deafness almost complete in both ears. Otoscopic findings: Right ear, membrane destroyed, no discharge. Left ear, membrane destroyed, large polyp from antrum region, scanty secretion. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 8 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 11 seconds. Galvanic reaction not taken. Caloric reaction, ice water for three minutes in left ear, mild rotatory nystagmus to the right. Remarks: Unsteady gait, moderate destruction of both acoustic and vestibular apparatus in both ears.

Case 54.—J. O., male, 13½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete

deafness both sides. Otoscopic findings, both sides normal. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 5 m.a., rotatory nystagmus to the right; anode 7 m.a., rotatory nystagmus to the left. Left ear, cathode 7 m.a., rotatory nystagmus to the left; anode 7 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf following meningitis at the age of one year.

Case 55.—A. J., male, 15½ years of age. No vertigo, tinnitus in the left, spontaneous nystagmus, slight rotatory nystagmus to the right when looking straight ahead. Hearing tests, complete deafness both sides. Otoscopic findings, both sides membrane normal. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 6 seconds; after ten turns to the right, with head inclined forward about 90 degrees, no nystagmus to the left. Galvanic reaction: Right ear, cathode 1 m.a., rotatory nystagmus to the right; anode 16 m.a., no reaction on the left. Left ear, cathode 12 m.a., no reaction on the left; anode 12 m.a., no reaction on the right. Caloric reaction not taken. Remarks: Deaf since five years of age, following meningitis.

Case 56.—M. B., female, 14½ years of age. No vertigo, tinnitus left side, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings: Membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 14 m.a., no reaction on the right; anode 14 m.a., no reaction on the left. Left ear, cathode 14 m.a., no reaction on the left; anode 14 m.a., no reaction on the right. Caloric reaction not taken. Remarks: Deaf since three years of age, following meningitis.

Case 57.—L. K., female, 9 years of age. No vertigo, tinnitus both sides, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings: Right ear, membrane slightly opaque. Left ear, normal. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right

ear, cathode 12 m.a., no reaction to the right; anode 12 m.a., no reaction to the left. Left ear, cathode 12 m.a., no reaction to the left; anode 12 m.a., no reaction to the right. Caloric reaction not taken. Remarks: Deaf since eight years of age, following meningitis.

Case 58.—S. P., female, 12½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membrane both sides opaque. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 12 m.a., no reaction to the right; anode 12 m.a., no reaction to the left. Left ear, cathode 12 m.a., no reaction to the left; anode 12 m.a., no reaction to the right. Caloric reaction not taken. Remarks: Deaf since two years of age, following meningitis.

Case 59.—M. W., female, 9½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, slight remains of hearing on both sides. Otoscopic findings, both sides normal membranes. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 12 m.a., no reaction to the right; anode 12 m.a., no reaction to the left. Left ear, cathode 12 m.a., no reaction to the left; anode 12 m.a., no reaction to the right. Caloric reaction not taken. Remarks: Deaf following meningitis.

Case 60.—M. G., male, 22 years of age. No vertigo, tinnitus both sides, more to the left, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membrane normal both sides. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 10 m.a., rotatory nystagmus to the right; anode 12 m.a., rotatory nystagmus to the left. Left ear, cathode 12 m.a., rotatory nystagmus to the left; anode 6 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since twelve years of age, following meningitis.

Case 61.—G. S., male, 10 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete

deafness both sides. Otoscopic findings, normal membranes both sides. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 12 m.a., no reaction to the right; anode 12 m.a., no reaction to the left. Left ear, cathode 12 m.a., no reaction to the left; anode 12 m.a., no reaction to the right. Caloric reaction not taken. Remarks: Deaf since five years of age, following a long fever. (Meningitis?)

Case 62.—N. K., male, 17 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Ostoscopic findings, both sides normal. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 18 m.a., no reaction to the right; anode 18 m.a., rotatory nystagmus to the left. Left ear, cathode 18 m.a., rotatory nystagmus to the left; anode 18 m.a., no reaction to the right. Caloric reaction not taken. Remarks: Deaf since birth.

Case 63.—A. N., male, 13½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing very greatly reduced both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 16 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 16 seconds. Galvanic reaction: Right ear, cathode 7 m.a., rotatory nystagmus to the right; anode 6 m.a., rotatory nystagmus to the left. Left ear, cathode 5 m.a., rotatory nystagmus to the left; anode 9 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since birth.

Case 64.—A. R., male, 15 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 25 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nys-

tagmus to the left 22 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Deaf since birth.

Case 65.—R. E., male, 14 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes opaque and retracted both sides. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 14 m.a., no reaction to the right; anode 14 m.a., no reaction to the left. Left ear, cathode 14 m.a., no reaction to the left; anode 14 m.a., no reaction to the right. Caloric reaction not taken. Remarks: Deaf since birth.

Case 66.—A. N., male, 13½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, both sides normal. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 10 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 10 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Deaf since birth.

Case 67.—O. F., female, 12½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 17 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 20 seconds. Galvanic reaction: Right ear, cathode 5 m.a., rotatory nystagmus to the right; anode 4 m.a., rotatory nystagmus to the left. Left ear, cathode 4 m.a., rotatory nystagmus to the left; anode 5 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since birth.

Case 68.—M. W., female, 15½ years of age. No vertigo, tinnitus both sides, no nystagmus. Hearing tests, very slight remains on both sides. Otoscopic findings, right membrane slightly opaque, left normal. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 20 seconds; after ten

turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 20 seconds. Galvanic reaction: Right ear, cathode 10 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 6 m.a., rotatory nystagmus to the left; anode 10 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since birth. Sister to O. W. (Case 69), also deaf since birth.

Case 69.—O. W., female, 14 years of age. No vertigo, tinnitus to the right, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 14 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 16 seconds. Galvanic reaction: Right ear, cathode 6 m.a., rotatory nystagmus to the right; anode 6 m.a., rotatory nystagmus to the left. Left ear, cathode 8 m.a., rotatory nystagmus to the left; anode 6 m. a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since birth. Sister to M. W. (Case 68), also deaf since birth.

Case 70.—A. S., male, 14½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membrane opaque both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 16 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 19 seconds. Galvanic reaction: Right ear, cathode 5 m.a., rotatory nystagmus to the right; anode 12 m.a., no reaction to the left. Left ear, cathode 12 m.a., no reaction to the left; anode 4 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since birth.

Case 71.—A. E., male, 11 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, slight remains of hearing. Otoscopic findings, membrane normal both sides. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 12 m.a., no reaction;

anode 12 m.a., no reaction. Left ear, cathode 12 m.a., no reaction; anode 12 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since birth.

Case 72.—V. H., male, 16½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, total destruction both membranes with granulations in tympanic cavity. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 8 m.a., rotatory nystagmus to the right; anode 11 m.a., rotatory nystagmus to the left. Left ear, cathode 8 m.a., rotatory nystagmus to the left; anode 8 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since two years of age, following measles.

Case 73.—A. W., female, 13½ years of age. No vertigo, no tinnitus to the left, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, total destruction of the membranes, both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 17 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 14 seconds. Galvanic reaction: Right ear, cathode 10 m.a., rotatory nystagmus to the right; anode 7 m.a., rotatory nystagmus to the left. Left ear, cathode 7 m.a., rotatory nystagmus to the left; anode 10 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since six years of age, following measles.

Case 74.—F. K., male, 13 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing greatly reduced on both sides. Otoscopic findings, membranes retracted and opaque both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 4 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 5 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 3 m.a., rotatory nystagmus to the left; anode 5 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Very deaf since three years of age from unknown cause.

Case 75.—M. B., male, 13½ years of age. No vertigo, no tinnitus, spontaneous nystagmus, slight rotatory nystagmus to the left when looking straight ahead. Hearing tests, very slight remains both sides. Otoscopic findings, membranes thickened and opaque both sides. After-turning nystagmus: After ten turns to the left, with head erect, and after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction, unable to examine because of nervous condition. Caloric reaction not taken. Remarks: Deaf since two years of age from a blow on the head.

Case 76.—K. K., male, 11½ years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, very greatly reduced in the right ear, complete deafness in the left. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 15 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 15 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 7 m.a., rotatory nystagmus to the left. Left ear, cathode 7 m.a., rotatory nystagmus to the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since five years of age, from a blow on the head.

Case 77.—J. B., male, 13½ years of age. No vertigo no tinnitus, no spontaneous nystagmus. Hearing very greatly reduced on both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 12 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 10 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 12 m.a., rotatory nystagmus to the left. Left ear, cathode 8 m.a., rotatory nystagmus to the left; anode 4 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Deaf since three years of age from a blow on the head.

Case 78.—L. A., male, 11 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, very slight remains both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory

nystagmus to the right 8 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 9 seconds. Galvanic reaction: Right ear, cathode 10 m.a., no reaction; anode 10 m.a., no reaction. Left ear, cathode 10 m.a., no reaction; anode 10 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since one year of age from a blow on the head.

Case 79.—F. L., male, 10 years of age. No vertigo, tinnitus both sides, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes slightly opaque and retracted both sides. After-turning nystagmus: After ten turns to the left, with head erect, and, after ten turns to the right, with head erect, no nystagmus, either to the right or to the left. Galvanic reaction: Right ear, cathode 10 m.a., no reaction; anode 10 m.a., no reaction. Left ear, cathode 10 m.a., no reaction; anode 10 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since eight years of age, following a fall on the back of the head.

Case 80.—M. L., male, 14 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head erect, and, after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 14 m.a., no reaction; anode 14 m.a., no reaction. Left ear, cathode 14 m.a., no reaction; anode 14 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since three years of age, following scarlet fever.

Case 81.—E. U., male, 16 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness both sides. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After ten turns to the left, with head erect, and, after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 12 m.a., no reaction; anode 12 m.a., no reaction. Left ear, cathode 12 m.a., no reaction; anode 12 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since three years of age, following typhoid fever.

Case 82.—H. N., female, 15 years of age. No vertigo, tinnitus to the left, no spontaneous nystagmus. Hearing tests,

very slight remains of hearing on both sides. Otoscopic findings, membranes normal on both sides. After-turning nystagmus: After ten tur. to the left, with head erect, and, after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 12 m.a., no reaction; anode 12 m.a., no reaction. Left ear, cathode 12 m.a., no reaction; anode 12 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since four years of age; cause unknown.

Case 83.—W. W., male, 11 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, complete deafness right side, slight remains on the left. Otoscopic findings, membranes opaque both sides, with chalk deposit on the right. After-turning nystagmus: After ten turns to the left, with head erect, and, after ten turns to the right, with head erect, no nystagmus either to the right or to the left. Galvanic reaction: Right ear, cathode 10 m.a., no reaction; anode 8 m.a., no reaction. Left ear, cathode 10 m.a., no reaction; anode 10 m.a., no reaction. Caloric reaction not taken. Remarks: Deaf since one year of age, following whooping cough.

Case 84.—J. E. T., male, 64 years of age. Vertigo present, probably of ocular origin, improved with correcting lens, has tinnitus, no spontaneous nystagmus. Hearing tests, marked reduction in both ears, more in the right. Otoscopic findings, both membranes slightly opaque and retracted. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 31 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 29 seconds. Galvanic reaction: Right ear, cathode 6 m.a., rotatory nystagmus to the right; anode 6 m.a., rotatory nystagmus to the left. Left ear, cathode 6 m.a., rotatory nystagmus to the left; anode 6 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Claims to have had cerebral hemorrhage eight years ago with vertigo, vomiting and complete loss of hearing.

Case 85.—D. M., male, 23 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, impairment of both sides, worse on the right. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings,

almost complete destruction of membrane on both sides with cholesteatoma and discharge. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 28 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 27 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 33 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 32 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 34 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 33 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 22 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 26 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Chronic middle ear suppuration both sides, fistula symptom positive left side.

Case 86.—J. T. D., female, 17 years of age. Vertigo pronounced, no tinnitus, spontaneous nystagmus horizontal to the right. Hearing tests, moderate reduction on the left. Weber to the left. Rinne negative to the left, right ear normal. Otoscopic findings, left ear, moderate destruction of membrane with discharge and granulations. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 5 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 14 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 17 seconds. Galvanic reaction: Right ear, cathode 6 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 5 m.a., rotatory nystagmus to the left; anode 6 m.a., rotatory nystagmus to the right. Caloric reaction: Cold water in left ear, head erect, rotatory nystagmus to the right. Remarks: Case of fistula of the external semicircular canal. Operated for radical mastoid, healing with retention of function in the vertical semicircular canal.

Case 87.—W. S., male, 56 years of age. Has vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, slight reduction both sides. Otoscopic findings, fairly normal membrane both sides. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 20 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 18 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 18 seconds. Galvanic reaction: Right ear, cathode 5 m.a., rotatory nystagmus to the right; anode 8 m.a., rotatory nystagmus to the left. Left ear, cathode 8 m.a., rotatory nystagmus to the left; anode 5 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Mild lesion of the eighth nerve, left side, of questionable origin.

Case 88.—J. A., male, 38 years of age. Vertigo present some months ago but not now, no tinnitus, spontaneous nystagmus to the right when looking straight ahead. Hearing tests, great reduction in both ears, more on the left. Weber to the left. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right membrane opaque and retracted. Left ear the same. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Injury to head one year ago.

Case 89.—W. H., male, 20 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, marked reduction on the right, left normal. Otoscopic findings, right canal narrowed, perforation of the membrane; impossible to get other anatomic details. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 20 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Acute mastoiditis. Operation.

Case 90.—J. B. D., male, 14 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, marked

reduction on the right side, left side normal. Otoscopic findings, extensive swelling over the right mastoid, canal narrowed, bulging of right membrane, pulsating secretion. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 10 seconds; after ten turns to the right, with head inclined forward about 90 degress, rotatory nystagmus to the left 24 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Acute mastoiditis right side.

Case 91.—D. M. B., male, 24 years of age. Vertigo present only by quick movements of the head, no tinnitus, no spontaneous nystagmus. Hearing tests, total deafness in left ear, right ear normal. Otoscopic findings, normal both sides. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 18 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 16 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 16 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 16 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Case of congenital deafness in left ear.

Case 92.—S. G., female, 52 years of age. Has vertigo, for the last three or four years much worse, has tinnitus, no spontaneous nystagmus. Hearing tests, marked reduction in both ears, more on the right. Otoscopic findings, membranes normal both sides. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 21 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 11 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Otosclerosis with internal ear involvement.

Case 93.—A. C., female, 43 years of age. No vertigo, tinnitus pronounced, no spontaneous nystagmus. Hearing tests, very marked reduction in both sides. Weber to the left. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings, membranes normal appearance both sides, promontory pinker than normal. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 11 seconds; after ten turns to the

right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 11 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Otosclerosis with involvement of inner ear.

Case 94.—A. J., male, 41 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, reduction of hearing in both ears, more on the right. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right canal narrowed, profuse offensive discharge, complete destruction of the membrane, attic granulations. Left ear, complete destruction of membrane, no secretion. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 19 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 17 seconds. Galvanic reaction not taken. Caloric reaction positive in the right ear. Remarks: Chronic middle ear suppuration with cholesteatoma right side. Chronic adhesive process following suppuration left side.

Case 95,—M. K., female, 13 years of age. Vertigo present, no tinnitus, spontaneous nystagmus rotatory to the right looking straight ahead. Hearing tests, marked impairment of hearing on right. Schwabach shortened on the right. Rinne negative on the right. Otoscopic findings, right ear, small perforation, profuse fetid pus. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 25 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 17 seconds. Galvanic reaction: Right ear, cathode 1 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 5 m.a., rotatory nystagmus to the left; anode 1 m.a., rotatory nystagmus to the right. Caloric reaction, cold water to right ear, rotatory nystagmus to the left. Remarks: Chronic middle ear suppuration on the right side, chronic adhesive process on the left.

Case 96.—E. C. T., female, 12 years of age. Has vertigo, tinnitus occasionally, no spontaneous nystagmus. Hearing tests, marked reduction both ears, more to the right. Weber to the left. Schwabach slightly lengthened on the right and

on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, large perforation, fetid purulent secretion, inspissated mucus on inner wall. Left ear, large perforation in superior posterior quadrant. Polyps in the antrum region. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 12 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 16 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Cholesteatoma in both ears.

Case 97.—K. F., female, 22 years of age. Has vertigo occasionally, no tinnitus, no spontaneous nystagmus. Hearing tests, marked reduction both ears, especially in right. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, complete destruction of membrane, profuse offensive discharge and cholesteatoma. Left ear, complete destruction of membrane, granulations on promontory, remnants of hammer handle adherent to the inner wall. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 23 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 22 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 17 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 3 m.a., rotatory nystagmus to the left. Left ear, cathode 3 m.a., rotatory nystagmus to the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction, apparently negative on left side, positive on the right. Remarks: Bilateral chronic middle ear suppuration.

Case 98.—F. S., male, 27 years of age. Vertigo has been present but description of it is poor, tinnitus both sides, no spontaneous nystagmus. Hearing tests, marked reduction. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, small part of membrane remaining, scant, dark, offensive discharge, ossicles gone, granulations present. Left ear, slight, offensive discharge, canal

full of polyps. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 9 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 6 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 16 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 20 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 8 seconds; after five turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 8 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 12 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 11 seconds. After fifteen turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 12 seconds; after fifteen turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 16 seconds. After twenty turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 16 seconds; after twenty turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 16 seconds. Galvanic reaction: Right ear, cathode 4 m.a., rotatory nystagmus to the right; anode 5½ m.a., rotatory nystagmus to the left. Left ear, cathode 4½ m.a., rotatory nystagmus to the left; anode 4½ m.a., rotatory nystagmus to the right. Caloric reaction: Cold water to the right ear, rotatory nystagmus to the left; cold water for three minutes in left ear, pronounced rotatory nystagmus to the right. Remarks: Bilateral chronic middle ear suppuration.

Case 99.—A. D., male, 33 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing reduced in the right ear. Schwabach lengthened on the right. Rinne negative on the right. Left ear normal. Otoscopic findings, right ear, membrane entirely destroyed, thick yellow, offensive discharge, cholesteatoma in the attic region. After-turning

nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 13 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 18 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 23 seconds. Galvanic reaction not taken. Caloric reaction: Right ear, positive. Remarks: Unilateral chronic middle ear suppuration.

Case 100.—L. U., male, 13 years of age. Vertigo present, tinnitus present, no spontaneous nystagmus. Hearing tests, marked reduction of the perceptive type. Otoscopic findings, offensive discharge in right ear. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 6 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 12 seconds. Galvanic reaction not taken. Caloric reaction: Cold water to the right ear negative. Remarks: Unilateral chronic middle ear suppuration with destruction of the labyrinth.

Case 101.—L. H., female, 34 years of age. Vertigo present with vomiting, no tinnitus, spontaneous nystagmus, rotatory to the right looking straight ahead. Hearing tests, great impairment of the right side. Weber to the right. Schwabach slightly shortened on the right. Rinne negative on the right. Otoscopic findings, membrane totally destroyed, hammer handle retracted, inner wall thick and red, secretion scant. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 52 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 20 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 15 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 7 m.a., rotatory nystagmus to the left. Left ear, cathode 7 m.a., rotatory nystagmus to the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction: Cold water to the right ear, rotatory nystagmus to the left. Remarks: Chronic middle ear suppuration right side, with acute congestion of the labyrinth.

Case 102.—H. E. F., male, 11 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, reduc-

tion both ears, worse in the left. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, large heart-shaped perforation, Shrapnell's membrane intact. Left ear, complete loss of membrane, hammer handle present and retracted, but not adherent to the inner wall, promontory pale, niche of the oval window visible. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 19 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 11 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Chronic middle ear suppuration for nine years.

Case 103.—M. P., female, 8 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, hearing very much reduced on left side; right side normal. Otoscopic findings, left ear, pulsating discharge; difficult to get anatomic details because of the swollen canal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 5 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 7 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 13 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 19 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Case of acute mastoiditis left side.

Case 104.—G. McM., male, 49 years of age. No vertigo, no tinnitus, spontaneous nystagmus horizontal to the right when looking straight ahead. Hearing tests, very marked reduction in hearing both sides. Weber to the right. Schwabach lengthened on the right and on the left. Rinne positive on the right and on the left. Stenger's test showed absolute deafness in left ear. Otoscopic findings: Right ear, membrane intact, picture of old catarrhal condition. Left ear, membrane intact. After-turning nystagmus: After ten turns to the

left, with head erect, horizontal nystagmus to the right 17 seconds; after ten turns to the right, with hear erect, horizontal nystagmus to the left 10 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 17 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 14 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Case of brain tumor about the cerebellar pontine angle, choked disc both sides.

Case 105.—W. H., male, 13 years of age. Has vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, marked reduction of hearing in the left ear. Weber to the right. Schwabach very short on the left. Rinne negative on the left. Otoscopic findings, left membrane torn, through which blood issued. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 12 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 11 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 16 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 20 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 16 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 24 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 17 seconds. After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 19 seconds; after five turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 7 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 17 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 15 seconds. After fifteen turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 24 seconds; after fifteen turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 18 seconds. Galvanic reaction: Right ear, cathode 3 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 5 m.a., rotatory nystagmus to the left; anode 3 m.a., rotatory nystagmus to the right. Caloric reaction: After prolonged

use of cold water to the left ear, rotatory nystagmus to the right, but not intensive. Remarks: Patient brought to the hospital unconscious, with blood running from left ear. Suspected basal fracture corroborated at time of operation by general sugeon, who unnecessarily removed the stapes. Was then referred to me, at time of partial destruction of both acoustic and vestibular functions.

Case 106.—E. E., female, 43 years of age. Has dizziness when making rapid turns, tinnitus slight in the left ear, no spontaneous nystagmus. Hearing tests, slightest reduction in left ear to low tones. Weber to the left. Otoscopic findings: Right ear, membrane normal. Left ear, small chalk deposit anterior inferior quadrant. After-turning nystagmus: After five turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 23 seconds; after five turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 20 seconds. Galvanic reaction: Right ear, cathode 5 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 5 m.a., rotatory nystagmus to the left; anode 5 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: From a neurologic standpoint, patient is a hypochondriac.

Case 107.—H. B. N., male, 43 years of age. Has vertigo, has tinnitus, no spontaneous nystagmus. Hearing tests, marked reduction in both ears, more in the right. Weber indifferent. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right canal filled with polyps; impossible to get anatomic details. Left membrane totally destroyed, polyps covering promontory. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 16 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Chronic middle ear suppuration both sides.

Case 108.—W. U. S., male, 29 years of age. Vertigo very slight, no tinnitus, no spontaneous nystagmus. Hearing tests, totally deaf in the left side; right side normal. Otoscopic findings, both membranes intact. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 19 seconds; after ten turns to the right, with

head erect, horizontal nystagmus to the left 16 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Injury to the left side of the head nine years ago.

Case 109.—E. M. J., female, 54 years of age. No vertigo, tinnitus more to the right side, no spontaneous nystagmus. Hearing tests, hearing reduced both sides. Weber indifferent. Schwabach shortened on the right and on the left. Rinne positive on the right and on the left side. Otoscopic findings, both membranes slightly opaque. After-turning nystagmus: After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 14 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 21 seconds. Galvanic reaction: Right ear, cathode 5 m.a., rotatory nystagmus to the right; anode 5 m.a., rotatory nystagmus to the left. Left ear, cathode 5 m.a., rotatory nystagmus to the left; anode 5 m.a., rotatory nystagmus to the right. Caloric reaction not taken. Remarks: Moderate degree of internal ear affection both sides, more pronounced on the right.

Case 110.—W. M., male, 75 years of age. Vertigo pronounced, no tinnitus, no spontaneous nystagmus. Hearing tests, reduction both ears, short of total on the left. Weber indifferent. Schwabach shortened on the right and on the left. Rinne positive on the right and negative on the left. Otoscopic findings, after removing cerumen, both membranes slightly retracted and opaque. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 16 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 15 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 15 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 12 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Patient had six degrees of exophoria.

Case 111.—M. B., male, 11 years of age. No vertigo, no tinnitus, no spontaneous nystagmus. Hearing tests, impaired on right side. Weber to the right. Schwabach lengthened on the right. Rinne negative on the right. Otoscopic findings, right membrane swollen, sagging of the posterior wall. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 14 seconds; after ten

turns to the right, with head erect, horizontal nystagmus to the left 21 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 14 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 20 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Acute mastoiditis right side.

Case 112.—W. L. D., male, 56 years of age. Vertigo present but not recently, no tinnitus, no spontaneous nystagmus. Hearing tests, normal by all tests. Otoscopic findings, after the removal of cerumen, both membranes normal. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 32 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 29 seconds. After eight turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 19 seconds; too sick to continue. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Blood pressure 200, transient vertigo, due probably to high blood pressure.

Case 113.—L. S., female, 16 years of age. Vertigo occasionally, no tinnitus, no spontaneous nystagmus. Hearing slightly reduced, more in the right ear. Weber to the right. Schwabach lengthened on the right and on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, newformed membrane, atrophic, depressed and adherent to inner wall, chalky deposit. Left ear, normal. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 10 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 8 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 15 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 13 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 21 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 18 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Nervous individual.

Case 114.—C. R., female, 35 years of age. Vertigo recently, tinnitus present, especially to the right, no spontaneous nystag-

mus. Hearing tests, marked impairment of hearing in both ears. Weber to the right. Schwabach normal on the right and slightly shortened on the left. Rinne negative on the right and on the left. Otoscopic findings: Right ear, complete destruction of membrane, remnant of hammer handle in contact with swollen, reddened inner wall, round firm polyp in attic antrum region, granulations in the hypotympanum. Left ear, membrane opaque, retracted, short process prominent. After-turning nystagmus: After five turns to the left, with head erect, horizontal nystagmus to the right 20 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 25 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 26 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 26 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 23 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 22 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 28 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 28 seconds. Galvanic reaction not taken. Caloric reaction: Patient claimed to be dizzy during the test, but no nystagmus was noticed. Remarks: Divergent strabismus, patient fixes with left eye.

Case 115.—F. O., male, 22 years of age. Vertigo for ten days; was in bed with grippe and was dizzy the entire time, tinnitus in right ear four or five weeks for the last six months, very loud noises, no spontaneous nystagmus. Hearing tests, slight impairment both sides. Weber to the left. Schwabach normal on the right and lengthened on the left. Rinne positive on the right and negative on the left. Otoscopic findings, right membrane red and retracted, chalk deposits. Left membrane opaque, red hammer handle, increased mobility. After-turning nystagmus. After five turns to the left, with head erect, horizontal nystagmus to the right 25 seconds; after five turns to the right, with head erect, horizontal nystagmus to the left 24 seconds. After ten turns to the left, with head erect, horizontal nystagmus to the right 35 seconds; after ten turns to the right, with head erect, horizontal nystagmus to

the left 33 seconds. After fifteen turns to the left, with head erect, horizontal nystagmus to the right 37 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 33 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 34 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 32 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Subacute otitis media both sides.

Case 116.—E. L. B., female, 21 years of age. Vertigo for two years, nauseated but does not vomit during attacks, tinnitus for two years in the right ear, no spontaneous nystagmus. Hearing normal on both sides. Otoscopic findings, posterior portion of the membrane slightly cloudy and overmobile, hammer handle normal both ears. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 28 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 28 seconds. After ten turns to the left, with head inclined forward about 90 degrees, rotatory nystagmus to the right 13 seconds; after ten turns to the right, with head inclined forward about 90 degrees, rotatory nystagmus to the left 13 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Vertigo was found to be of ocular origin.

Case 117.—S. P., female, 23 years of age. Vertigo occasionally, no tinnitus, no spontaneous nystagmus. Hearing tests, normal both sides. Otoscopic findings: Right ear, large chalk deposits posterior quadrant, and smaller one in anterior quadrant. Left ear, membrane atrophic, chorda tympani nerve seen. After-turning nystagmus: After ten turns to the left, with head erect, horizontal nystagmus to the right 34 seconds; after ten turns to the right, with head erect, horizontal nystagmus to the left 33 seconds. After fifteen turns to the left with head erect, horizontal nystagmus to the right 38 seconds; after fifteen turns to the right, with head erect, horizontal nystagmus to the left 38 seconds. After twenty turns to the left, with head erect, horizontal nystagmus to the right 40 seconds; after twenty turns to the right, with head erect, horizontal nystagmus to the left 39 seconds. Galvanic reaction not taken. Caloric reaction not taken. Remarks: Despite the fact that both membranes presented abnormal appearances, the hearing was normal both sides.

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XXVI.

VERTIGO: ITS CAUSES AND METHODS OF DIAGNOSIS.*

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The subject of vertigo is as old as the healing art itself, and medical literature abounds with essays on the subject. When vertigo was seen to accompany certain diseases, it was spoken of as vertigo belonging to that affection. Thus, when it appeared in the course of nephritis, it was kidney vertigo; if in association with an affection of the liver, it was hepatic vertigo, and so on. As a result, there were almost as many kinds of vertigo as there are diseases; but when the vertigo appeared independently and not in the course of some disease, it was termed idiopathic or essential vertigo. That vertigo was essentially a disturbance of equilibration was gleaned long ago, but so long as the equilibratory function itself was not clearly understood, vertigo remained in the land of mystery.

For many centuries the individual was regarded as possessing only five special senses; that is, sight, hearing, taste, smell, and touch. A great advance was made when the sixth sense —the socalled "muscle-joint-and-splanchnic sense"—came to be recognized. The recent studies of the internal ear, however, show us that there is still another, seventh special sense —the socalled "static sense." This static sense is a special sense as truly as any of the others, and corresponds to them in its general anatomic and physiologic requirements. Similar to them, it has an "end organ" for receiving stimuli, a conducting nerve for conveying sensations, and definite nerve tracts leading to brain centers where the stimuli are interpreted and translated into equilibration. A host of investi-

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gators have conducted experiments showing the effect on equilibration when the internal ear is either partly or completely destroyed. As a result of these, it is now universally recognized that the static portion of the internal ear is the chief organ of equilibration—in other words, it is the "end organ" of this seventh "static sense." To be sure, the ear is not the only organ concerned with the equilibratory function. The equilibrium of the body is maintained because of a harmonious cooperation of three senses—sight, muscle, and static sense. Of the three, however, the end organ of the static sense is of peculiar interest, because its only function is the maintenance of balance, whereas with the other two, equilibration is simply a collateral function.

Summing up, then, we must have in mind the following:

1. Equilibrium of the body is maintained chiefly by the ear and its associated pathways and centers known as the vestibular apparatus aided by the sense of sight and the muscle sense.

2. Vertigo is a conscious sensation perceived within the brain when perfect equilibration is interfered with.

This makes it evident that in considering the causes of vertigo, we must look for those disturbances which will affect the chief organ of equilibration—that is, the vestibular apparatus. From a medical standpoint, therefore, the most important aspect of the study of the static portion of the internal ear and its innervation is the subject of vertigo. The doctor is often confronted with cases of dizziness that tax his ingenuity to the utmost, and it is surely not overstating the fact to say that at best he can only guess as to its significance. The realization that the ear is the chief organ of balance throws a new light on its importance. Previous to the conception of the ear as an equilibratory organ, the general practitioner regarded the ear somewhat as follows: If the patient is deaf, a specialist should see what he can do to improve the hearing, and if there is any inflammation of the ear, it should receive local attention. But far more important, and yet scarcely recognized, is the part that the static portion of the ear plays in the well-being of the entire body. The aurist naturally knows of the recent advances in the study of the ear, but it might not be amiss to emphasize their importance to the general practitioner as well, because of:

1. The necessity of recognizing internal ear conditions which he would overlook if he were not acquainted with the subject.

2. The far-reaching significance of the ear as the sense organ of equilibration.

3. The value of the new ear tests in determining the cause of vertigo, no matter what its type or origin. It is with such cases that the physician comes in daily contact.

The general practitioner is familiar with conditions of the external ear, the middle ear, and the cochlear portion of the internal ear. In the external ear he encounters impacted cerumen, foreign bodies, eczema, and furunculosis. In the middle ear he has to deal with various types of deafness and with inflammatory conditions. Affections of the cochlea manifect themselves by impaired hearing and noises in the head. The attention of the physician, therefore, is immediately called to the ear—in fact, the patient himself recognizes that he has an ear condition. But whereas all these are of interest from a local standpoint only, the static portion of the ear, on the contrary, is part and parcel and servant of the whole organism. A big, strong, healthy man is unable to stand up or even sit up in bed because of a slight congestion of the internal ear; perhaps the pathologic process is no larger in extent than the head of a pin, and yet the man is entirely incapacitated. Just as affections of the cochlea are manifested by impaired hearing and tinnitus, the disturbance of the static portion of the internal ear produces dizziness, staggering, nausea, vomiting, and diarrhea; but whereas tinnitus and deafness at once point to the ear, we venture to say that when confronted with dizziness and nausea, the general practitioner is not apt to suspect the ear. The cases of vertigo from ear conditions with nausea, vomiting, and retching, that are treated with "stomach powders" are legion; and it cannot be too strongly emphasized that vertigo from whatever cause can result only from an affection of the vestibular apparatus directly, so that the condition of the ear and its associated pathways should be thoroughly investigated.

Vertigo may be of two kinds:

1. Experimental or induced vertigo.
2. Vertigo resulting from disease.

Experimental or induced vertigo results when the equilibratory end organ is powerfully stimulated by artificial means. Normally every conceivable motion of the body—be it in a linear or rotary plane—causes a movement of the lymph in some portion of the static labyrinth, either in the saccule or utricle, or in the semicircular canals, which in turn stimulates the sensitive hair cells of the end organs placed there. The impulses thus generated are carried along the vestibular nerve pathways to their respective centers. Inherited memory dating back for countless ages, as well as innumerable repetitions of this sort during the individual's lifetime, result in a correct interpretation as to the meaning of all these stimuli, and the person learns to know that a certain stimulus or set of stimuli always means a certain kind of motion. The individual has also learned that this sort of information is reliable and true and in accordance with fact. He is, therefore, not in the least disturbed by them. Should the lymph in the labyrinth, however, be made to move violently by artificial means, such as can be done by turning an individual in a revolving chair or douching the ear with cold or hot water, he would then be getting information which is not true to fact. For example, after he is turned in a chair and suddenly stopped, the endolymph still continues circulating, conveying the information to the brain that he is moving, whereas, as a matter of fact, he is not. Here, then, he has information which is not true to fact, and he therefore has vertigo. While he was being turned, the endolymph also moved and conveyed to him the sensation of motion, but that produced no vertigo because it was in accordance with actual facts—his previous experience assured him that he actually was moving. Cooling or heating a portion of the labyrinth by means of douching also produces motion of the lymph. Since, however, the individual is sitting quietly in the chair all this time, such information is not in accordance with fact, and therefore results in vertigo. This induced or experimental vertigo is of great service in the study of the integrity of the pathways responsible for this vertigo, and can be made of great clinical value.

Vertigo resulting from disease is entirely different. It results whenever there is an interference with the perfect balance between the special static organs on each side. Both

ears always act in unison, and are constantly sending out an equal flow of impulses to the musculature of the body, insuring perfect equilibration. When disease destroys one side completely or partially, or produces an irritation—be it in the ear itself or along any pathway leading from it—there immediately results an inequality of tonic influence between the two sides, with the resulting disturbance in equilibration and vertigo. The important thing to remember is that it is always a disturbance of the ear and its associated pathways that can result in vertigo, and nothing else. When remote organs produce vertigo, it is only because they definitely influence the ear mechanism.

Vertigo may be caused by:

1. Lesions within the internal ear, such as labyrinthitis of the various types, or effusions or hemorrhage within the internal ear. Inflammatory conditions of the middle ear, for instance, acute otitis media or mild inflammation of the inner ear itself, produce only irritative effects, if any, so that the patient suffers from more or less vertigo as long as the acute state of the congestion lasts, the vertigo vanishing with the disappearance of the inflammation. On the other hand, slow degenerative changes within the labyrinth, or sudden destruction of the whole or part of one labyrinth, are accompanied by marked vertigo, nausea, and vomiting, and loss of equilibration. Sudden destruction of the whole or part of the labyrinth may be produced, rarely by trauma, but usually by hemorrhage or serous effusion into it, and it may occur in diabetes, Bright's disease, or in any condition where the vascular system becomes affected. It is this class of cases that exhibits the socalled Meniere's symptom complex. The hearing in these cases is usually markedly affected, if not altogether gone, and the condition is characterized by repeated attacks with a suddenness of onset, the violence of the symptoms quickly reaching a climax and then gradually subsiding, and all of it disappearing when the brain centers have learned to compensate.

2. Toxemias, affecting the ear or other portion of the vestibular apparatus, such as ptomain poisoning, constipation, alcoholism, poisoning by chemicals, as, for example, lead poisoning, nephritis, gout, rheumatism, syphilis, and the toxemia of infectious fevers, such as scarlatina, typhoid fever, and mumps.

3. Definite lesions along the pathways from the ear within the brain itself, such as tumor, hemorrhage, thrombus, infarct, abscess, gumma, tubercle, specific neuritis, multiple sclerosis, syringomyelia, polioencephalitis, meningitis, or ocular conditions. Here, disturbances in the visual mechanism, reflexly affect the vestibular apparatus.

If vertigo can be produced only by disease affecting the vestibular apparatus, then it follows that in order to diagnosticate the cause of vertigo, we must employ those tests which analyze the function of this apparatus. Such an examination consists of a search for any spontaneous vestibular phenomena which may be present, as well as an analysis of the responses to ear stimulation. Of the two methods, the latter is by far the more important. We start out with a fixed and definite idea that a normal internal ear with intact nerve pathways leading to normal nerve centers will invariably produce a rhythmic nystagmus and vertigo when a stimulus is applied. The nystagmus lasts twenty-six seconds in a certain direction when the individual is turned in a chair ten times, and the vertigo is also of twenty-six seconds' duration. Because of the vertigo there also appear past-pointing and falling. If, on the other hand, stimulation of the internal ear fails to produce one or all of the responses, it is positive evidence of an impairment along some point of this apparatus. A knowledge of the anatomy of this vestibular mechanism is, of course, indispensable, and one's ability to make a correct diagnosis as to the location of any lesion causing vertigo depends entirely as to how well he can visualize the anatomic pathways which constitute this vestibular apparatus as far as those pathways are known. The internal ear is the starting point for all impulses. Any form of stimulation applied to the internal ear, be it turning or douching, affects all nerve endings in like manner, and we think it highly improbable that there could be a lesion that would affect the hair cells concerned in the production of nystagmus without affecting also the hair cells concerned in the production of vertigo. If, therefore, stimulation of the internal ear fails to produce any response at all, or when all the responses are affected similarly, we conclude that the lesion is either in the labyrinth itself, or in the eighth nerve. If, on the other hand, stimulation of the internal ear produces one good

reaction at the same time that the other responses are absent or markedly impaired, it is obvious that the internal ear itself and the eighth nerve are intact, and that the lesion is located centrally; that is, after the eighth nerve has divided into its component portions on entering the brain stem at the junction of the medulla oblongata and pons. If the nystagmus is impaired, the block is obviously in the vestibuloocular tracts; whereas, if it is the vertigo that is impaired, the block must be located along the vestibulocerebellocerebral tracts—those concerned with the production of vertigo. A knowledge of the course which all these tracts pursue within the brain substance enables one to locate accurately the site of the lesion responsible for the vertigo. However, it not infrequently happens that patients complaining of vertigo, when tested show normal reactions to ear stimulation. In these cases it is obvious that we are dealing with a condition where the vestibular apparatus is irritated at some point, and a search should be made for such a source of irritation. This may be a visual defect or a focal infection in the tonsils, teeth, stomach, kidneys, etc.

Therefore, just as we examine the urine in suspected cases of nephritis or diabetes, or as we have a Wassermann test made in suspected cases of syphilis, just so the ear tests enable us to analyze the apparatus responsible for dizziness. Instead of guessing, we then know the reason for the vertigo. Based on an experience of hundreds of examinations of pathologic cases in the Department of Neuro-Otology at the University of Pennsylvania, we do not hesitate to say that it is very rare that a "dizzy" case remains obscure after these ear tests, and in most instances the diagnosis becomes clear and simple. After these tests are made we are able to say, for example, that the patient is dizzy because of a toxic impairment of the labyrinth, a labyrinthitis, an organic lesion in some part of the vestibular tract, such as is produced, for example, by brain tumor, in internal hydrocephalus, brain abscess, or multiple sclerosis; or in the presence of normal reactions, we can state with assurance that the vertigo is purely functional or due to an evanescent toxin.

In conclusion, the following points may be emphasized:

1. There can be no vertigo unless there is a disturbance of the vestibular apparatus. When diseases in remote organs are

accompanied by vertigo, it is because those pathologic states somehow affect the vestibular apparatus.

2. Vertigo may be due to some simple irritation of the vestibular tracts. In such a case they are temporary and fleeting in character, leaving the apparatus itself intact, and the tests will therefore show normal responses.

3. There may be a lesion of the internal ear itself.

4. There may be a lesion situated within the brain along some pathway in association with the ear.

Disturbance of the vestibular apparatus with vertigo can be definitely analyzed and diagnosed by means of the ear tests.

XXVII.

CONCERNING THE USE OF THE ELECTRIC CAUTERY IN THE NOSE.

By William A. Hitschler, M. D.,

Philadelphia.

An article in a recent number of the Annals of Otology, Rhinology and Laryngology condemns almost unreservedly the use of the electric cautery in the nose. Evidently, rhinologists are not all of the same mind on the subject.

When this therapeutic measure was first introduced, it naturally had as many advocates, both wise and foolish, as any other new therapeutic measure. After it had been used, and also abused, for a while, it naturally had as many adverse critics, both wise and foolish, as any other new therapeutic measure. Whether it is the sewing of a button on milady's dress or her jejunum, the pendulum will not be denied its swing. And there is a philosophy in the oscillation of the pendulum.

In contemplating the various movements of medical thought and action we cannot help being reminded of one of Pudd'nhead Wilson's maxims. To quote from Mark Twain: "We should be careful to get out of an experience only the wisdom that is in it—and stop there; lest we be like the cat that sits down on a hot stove lid. She will never sit down on a hot stove lid again—and that is well; but she will never sit down on a cold one any more."

Impulsiveness is frequently a characteristic of medical human nature. It is closely associated with enthusiasm. The one is to be condemned and the other commended. Too often, however, no distinction is observed, and doubt and hesitation exact their toll. Hence the swing of the pendulum, back and forth, and the truth, situated at either end of the arc, or at some point between, is discovered only by the philosophy

embodied in the above quoted maxim. The lessons of history, of experience, too often pass unheeded.

Harm can come of almost any therapeutic measure, and the electric cautery is no exception. But it is hardly fair to deny, at the classic one fell swoop, the usefulness of the cautery when properly applied. For it is a most efficient weapon in the warfare against some very common nasal diseases.

There may be a dispute as to what constitutes the proper application of the cautery, but the conflicting opinions on the subject will eventually disclose the truth. A little more circumspection, a little more reflection, and a wide divergence may blend into an harmonious unanimity of opinion which cannot be used as a shuttlecock by the animadversively inclined.

The electric cautery is a most useful agent for combating certain nasal conditions, and when properly done and the wound subsequently treated, is attended by no harmful after-effects. When too extensive an area is cauterized, and when subsequent treatment is curtailed or omitted, the stage may be set for pernicious sequelæ, such as persistent crusting and adhesions; and these constitute the chief objections to the abuse of the cautery, but not to its use. Temporary crusting always occurs, and continues for varying periods of time—a few weeks or a couple of months at the most. I cannot recall, at the present moment, a single case of permanent crusting. And in eliciting the symptoms of the patient in this particular, we should keep in mind the common crusting at Kiesselbach's spot, which may bias our conclusions if no note of it has been made previously. And here is the point. At Kiesselbach's spot the metamorphosed epithelium has two dimensions, length and breadth, while after a linear cauterization it has relatively but one dimension—length. The mucous membrane finds it easier to grow over a line than over an area. In many cases, after a comparatively short lapse of time, it is most difficult to determine, by the closest inspection, whether or not the cautery has been used. This applies particularly to those cases of simple vasomotor paresis without hypertrophy. Where hypertrophy or hyperplasia has been present, a linear depression or furrow may be visible; but as that is one of the results sought for in such cases, it is simply evidence that the cauteri-

zation has accomplished its purpose. And it is well within the limits of truth, supported by clinical experience, that linear cauterization practically removes the objection to crusting. When the nasal fossa is narrow, or when two opposite surfaces are cauterized at the same time, adhesions may result. In the former case, ordinary after-treatment, continued until the wound has healed, will serve as a preventive. And there is no reasonable excuse for simultaneous cauterization of opposing surfaces. Even when done accidentally, a little care and persistence in the after-treatment will obviate all difficulty.

The inflammatory reaction which follows the use of the cautery is occasionally pronounced, and in some cases—so it has been stated—has led to meningitis. Pneumonia, too, has followed the administration of ether as an anesthetic; but the careful anesthetist will be reasonably sure that no inflammation of the respiratory passages is present when the ether is administered. And the careful rhinologist will be reasonably sure that there is no active inflammation of the nasal mucosa present when he uses the cautery; and, in addition, he will cleanse the nasal fossæ before beginning the operation, and also avoid the olfactory region.

The subsequent inflammatory reaction cannot be seriously considered an argument against the use of the cautery. It is often mild; and even when severe, gives the patient no troublesome symptom excepting a temporary nasal obstruction, to which, indeed, he has already been subjected.

The alleged evil after-effects above mentioned—towit: persistent crusting, adhesions and excessive inflammatory reaction —are not inevitable sequelæ, and are likely due to errors of judgment and technic, or neglect of after-treatment.

It would serve no purpose to attempt an enumeration, to a body of rhinologists, of all the indications for the use of the electric cautery in the nose. But there are several types of cases which, in themselves, provide sufficient justification for its employment. First, those cases characterized by various degrees of vasomotor paresis of the turbinated bodies, especially the inferior, giving rise to alternate, intermittent, nocturnal or decubital nasal obstructions; second, cases of chronic rhinitis not secondary to some other nasal condition, and characterized by a thick mucous or mucopurulent discharge, with

or without beginning hypertrophy; and third, cases of reflex irritation—e. g., asthma, the vasomotor rhinitis group, nocturnal enuresis, dysmenorrhea, etc.

Other methods of treatment are applicable, of course, in such cases. But there are times when other treatment fails, and it often happens that cauterization is attended by results which can only be described as brilliant.

XXVIII.

FACIAL PARALYSIS AS A COMPLICATION OF ACUTE OTITIS MEDIA.

By Edward Barton Jones, M. D.,

Washington, D. C.

Facial paralysis is not uncommon as a sequel of chronic suppuration in the ear, but its appearance in connection with acute otitis media does not seem to be of very frequent occurrence. On looking over the literature of the past ten years, I find that some of the books on otology do not mention it at all, and that some others give it scant notice. I found very few actual cases reported. There is a wide divergence of opinion as to its frequency and significance.

To explain the involvement of the facial nerve in an acute inflammation of the middle ear, reference must be made to their anatomic relationship. The fallopian canal is separated from the tympanum by a thin wall of bone, sometimes extremely thin. Often there is a dehiscence in this wall, usually situated just above the oval window, and the nerve, therefore, in such instances, lies behind the mucous membrane lining the cavity of the middle ear. There are other openings in the bony wall between the canal and the cavity, such as those for the passage of the chorda tympani nerve, the branch from the facial to the stapedius muscle, and the tympanic branch of the stylomastoid artery. It is believed that these offer channels for the spread of the inflammation. The impairment of the function of the nerve is brought about either by direct involvement, the inflammation extending to its sheath, or by pressure of exudation within its canal, or, in the opinion of some authors, by pressure from the tympanum. Ulceration and caries of the wall may take place, even in the acute stage.

The existence of the dehiscence is held by most writers to be the probable explanation of the way in which the facial

nerve becomes affected, either as permitting pressure upon the nerve by the pent-up exudate in the tympanum, or as providing a favorable opening for the extension of the inflammation. It is difficult to see how an exudate in the tympanum pressing against an intact mucous membrane covering such a deficiency in the bone could exert sufficient force to produce a paralysis. It would be more reasonable, perhaps, to expect such an effect if the mucous membrane were intensely swollen and projected into the canal. If the paralysis were due solely to pressure upon the nerve from fluid in the tympanum, should it not immediately, or almost immediately, disappear upon relief of tension through the drumhead?

We are familiar with the fact that perforation of the drumhead may occur in a very early stage of otitis media and before the formation of pus. It is presumably a stronger wall, although of greater area, than the mucous membrane overlying the deficiency in the bone, and it may not be unreasonable to assume that the same process may take place in the latter with even greater rapidity and consequent exposure of the nerve to the action of the inflammatory exudate containing the exciting organisms.

Politzer says that whether a dehiscence in the fallopian canal favors the development of facial paralysis has not been proved; a fact speaking against it is that in caries and necrosis of the walls of the cavum tympani the facial nerve has sometimes been found lying free in the tympanic cavity completely surrounded by pus, without any symptoms of facial paralysis ever having been observed during the life of the patient. It might be inferred from this observation that to produce paralysis of the nerve (from middle ear disease) it must be subjected to pressure, such as could be exerted upon it while confined within its narrow channel.

The diagnosis is obvious. The paralysis may come on suddenly and be quite complete in a few hours, or it may be more gradual in its appearance, and certain muscles may be more affected than others. Severe pain in the ear and side of the face may precede its development; in other cases no pain is experienced, and the paralysis is ushered in by twitchings of the muscles. If the lesion of the nerve is limited in extent, its site in the canal can be determined, it is said, as follows:

If above the point where the chorda tympani takes its departure and below the branch to the stapedius muscle, the sense of taste in the anterior third of the tongue on the same side is disturbed. If above the twig to the stapedius there is, in addition, severe tinnitus and increased sensitiveness to low tones. If above the geniculate ganglion the sense of taste is not disturbed. There is alteration also in the secretion of saliva when the chorda tympani is involved.

The prognosis, as a rule, is good; a favorable outcome may be usually anticipated; however, recovery may be extremely slow, and in some instances permanent impairment may be the result.

The treatment of this condition is primarily that of the ear disease. If the middle ear inflammation receives proper attention the paralysis will usually disappear more or less promptly. Free drainage from the tympanum must be maintained; the membrane should be incised, if there is not already a perforation sufficiently large for escape of the exudate; if an existing perforation is too small for ready exit, it should be enlarged. In the absence of definite mastoid signs, operation does not seem to be indicated. Some authors recommend counterirritation over the mastoid and the internal administration of iodid of potassium. If the paralysis should persist in spite of improvement in the middle ear, electricity and massage should be employed.

This complication seems to be sufficiently rare to warrant the report of a case that came under my observation.

The patient was a boy, five years old. He had a history of glandular tuberculosis, particularly of the mesenteric glands, for two or more years, but seemingly had recovered from this disease. He had had chicken pox and a moderate influenza in the second week of February, 1915, but appeared to be entirely well during the next three weeks. His physician was again called to see him on March 8th, the boy having been awake all night with earache. On examination the ear appeared normal, and he was apparently well next day. The following day he again complained of pain; the membrane was pink, but not bulging, and its condition was the same on the 11th, and presented little change on the 12th, the paralysis be-

ginning in the morning of that day. He had a temperature of 101°.

I first saw him on March 13th. The paralysis was almost complete. The membrane was red and bulging; on incision there was a serous bloody discharge, but no pus. A culture of the exudate showed a pure streptococcus of unusual variety. An autogenous vaccine was directed to be prepared.

March 17th. Paralysis very noticeably less. Temperature for several days has not been above 100°, and much of the time normal.

March 20th. Temperature, 102.6°; he felt sick.

March 21st. Paralysis practically gone. Temperature, normal. This day the vaccine was first ready for use and he was given a dose of five million.

March 23d. Cervical glands enlarged; no tenderness over mastoid.

March 24th. Temperature normal in morning; at noon a sudden pain in the ear, and temperature 101°. He received a dose of vaccine, seventy-five million. In the afternoon there was some swelling in front and below the ear; no appreciable tenderness over mastoid. Much redness and swelling in the upper half of the drum membrane, extending on to the meatal wall. Temperature 103° in the evening. Next day he felt very well; temperature near normal, swelling gone and membrane looking better. From this date the temperature was practically normal. He received four more injections of vaccine, at intervals of about three days, in increasing dosage, the last being five hundred million on April 8th, the date of last record of any discharge from the ear. The patient was then completely recovered.

During the course of the disease the meatus was kept clear by irrigation at suitable intervals. The ear drained well and the exudate maintained a serous character during two weeks, becoming slightly mucopurulent as it lessened in quantity, and ceasing in about four weeks from the date of the incision. The paralysis lasted about ten days. Whether or not the vaccine had any effect on the middle ear inflammation, it had no influence over the duration of the paralysis, as its use was not begun until the latter was ending.

XXIX.

PRIMARY JUGULAR THROMBOSIS DUE TO TONSIL INFECTION.*

By Charles Goodman, M. D.,

New York.

On December 8th a married woman, thirty-five years of age, was admitted to the Beth Israel Hospital. When five years of age she had had typhoid fever.

She had received treatment for follicular tonsillitis for ten days before her admission to the hospital. Five days previous she had had two or three chills, followed by fever and by profuse perspiration, accompanied with vomiting. A tender swelling was noticed on the left side of her neck, and she was referred to the hospital. Upon admission she had a temperature of 104°. She had a severe chill which was followed by a rise of temperature to 107.2°. The patient became delirious. The blood culture taken on the day of admission and again on the following day proved to be negative. Her blood count was 12,600, polynuclears eighty-four per cent. An examination made by Dr. Kopetsky of the ears, throat and accessory sinuses was negative.

On the evening of December 8th the patient presented an induration extending along the anterior border of the sternocleidomastoid on the left side. The patient appeared to be very ill and septic.

I confirmed the diagnosis made by Dr. Schneider, the house physician, of a probable septic thrombophlebitis; the chills, the hyperpyrexia and delirium indicating immediate operation. The pharynx was markedly congested, otherwise presented nothing abnormal.

The internal jugular vein was exposed by the usual incision

*This patient was presented before the Surgical Section of the New York Academy of Medicine, at a stated meeting held January 5, 1917.

along the anterior border of the sternocleidomastoid muscle. It was found surrounded by considerable infiltration of the tissues. The vein was exposed throughout its entire length and ligated near the clavicle. Thrombosis was situated at and included part of the facial tributary. The vein was resected from the clavicle below with a portion of the facial tributary to the jugular foramen. The greater portion of the wound was sutured and a drain was inserted in either end. The temperature gradually fell and on the fifth day was normal. The patient was out of bed on the fourteenth day.

During the operation the question arose as to the advisability of exposing the sinus, because I did not get blood readily with the aspirating needle introduced into the upper part of the jugular. I maintained that the danger of dissemination of infectious material throughout the body had been eliminated with the ligation of the jugular vein below the apparent thrombosis before the resection of the vein was undertaken. The exposure of the sinus was, therefore, not made, as the chiseling necessary for its exposure would have diminished the chances of the ultimate recovery of the patient.

Up to the present time there has been no indication for any further operative procedure.

The interesting features of this case are the direct infection of the jugular by the lymphatic route from the tonsils and the pharynx instead of the usual primary involvement of the sinus, and a secondary extension to the jugular complicating mastoiditis.

In recent literature, Long's case of sore throat with streptococcic invasion of the internal jugular vein and subsequent thrombi most nearly approaches this one.

Barnes, in a compilation of reports of systemic infections secondary to follicular tonsillitis involving the pharyngeal wall, mentions no direct infection of jugular vein as a complication.

We may conclude that the probable bacterial agent was streptococcus, since it is the most frequent factor in acute infectious processes in the mouth and throat (Braun) as well as in thrombosis.

It is interesting to quote here the explanation given by Libman and Celler of the negative blood cultures they ob-

tained in a number of cases when sinus thrombosis of infectious origin was present: "We are inclined to believe that there may be early no bacteremia, that the bacteria may then be found perhaps in increasing number, and that it is possible for the bacteria to decrease in number and even to disappear before there has been any operative interference. It is also possible that in certain cases there is below the infected clot an obturating, noninfected clot which prevents the bacteria from entering the circulation. We can also imagine the possibility of there being an infected clot which completely closes the vessel. Another possible explanation is that in the course of the infection the blood may acquire a higher bactericidal power."

By analogy the same arguments might be applied to the negative blood cultures in our case of jugular thrombosis we have just reported.

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XXX.

TRANSILLUMINATION OF THE LARYNX AND UPPER TRACHEA.*

(FIRST COMMUNICATION.)

By Frank R. Spencer, A. B., M. D.,

Boulder.

In June, 1916, while the guest of Dr. Joseph C. Beck, at the North Chicago Hospital, I was invited to look into the larynx and trachea of a patient suspended with Lynch's suspension laryngoscope. The Beck light, on a stand, was at the patient's left shoulder, and the shade so placed that much of the light was thrown on the left side of the larynx and upper trachea. I chanced to view the interior of the larynx and trachea the instant before my head mirror could throw the light into the upper respiratory tract, and I had my attention immediately attracted by the readiness with which the interior was illuminated by the light on the stand. I called Dr. Beck's attention to this, and the word "transillumination" quickly suggested itself to his active mind. The thought that I had discovered something new occurred to me, but upon looking up the literature I found I was mistaken.

Whether this particular method of transillumination is of any real value, and whether this method of using it is original, I cannot say, but I think it is. However, transillumination of the larynx was first practiced by Czermak,[4] but was later perfected by Voltolini.[6] Freudenthal states in a letter that he has had experience with transillumination of the larynx, and that he was the first one in the United States to demonstrate the method. He has written articles upon this in German[2] and in English.[1 3]

I shall present this method for what it is worth and let the

*Read before the Western Section of the American Laryngological, Rhinological and Otological Society, at Colorado Springs, March 17, 1917.

future decide its real merit and permanent place in laryngology. However, I am afraid transillumination of the larynx and trachea has only very limited practical value when compared with transillumination of the maxillary sinuses, because it is so easy to obtain a good view of the interior of the former and so difficult to do so with the latter.

The following are the methods of applying transillumination to the larynx:

1. Voltolini[6] used a metal covered electric light for condensing the rays of light on the exterior of the larynx.

2. Freudenthal[2] improved upon Voltolini's model of laryngeal transilluminator and applied it either at the pomum Adami or near the cricoid cartilage, and introduced the heated laryngeal mirror into the dark pharynx so as to obtain a good view.

3. Cheatham has used a de Zeng transilluminator, but he does not say just how he applied this. Several other laryngologists have used this test, or method of examination, in different ways, judging from their replies to my circular letter of inquiry to all the fellows of this society.

4. The Beck light, on a stand, may be used to good advantage to light the larynx while the patient is suspended, as I mentioned in the first part of this paper. Whatever the method of applying this, the principle is the same.

Transillumination, even in normal patients, should be of value for inspection of the larynx and upper trachea. In cases of benign growths it should give a fairly definite idea of the amount of inflammatory thickening about the point of attachment. This will be of even greater value in cases of malignancy or suspected malignancy. The same may be said of tuberculous or syphilitic ulcers, lupus, chondritis, perichondritis, etc. Freudenthal in a personal communication relates the following: "I recollect one case which I saw, together with Dr. Lefferts and the late Dr. Asch. It was a man with terrific stridor. Both these colleagues had made the diagnosis of aneurism, while my diagnosis was carcinoma of the trachea. A dark shadow was plainly seen on transillumination. At the autopsy both conditions were found—i. e., a cancer, on top of which (or on the side, I do not recollect particulars) was an aneurism." As this was prior to the bronchoscopic and sus-

pension laryngoscopic era in medicine, such a test was of more value then than now, other things being equal.

As the test can be made during either suspension or direct laryngoscopy, or by a modification of indirect laryngoscopy, it cannot be used as easily as transillumination of the maxillary sinuses, for instance. Direct or indirect inspection of the mucosa of the upper respiratory tract, by any of the standard methods, is of greater value than transillumination, especially since indirect inspection is comparatively easy. On the other hand, we should use in laryngology, as in the other specialties, all the tests at our disposal, if we cultivate accuracy in diagnosis. The average case may not and probably will not require it, but the difficult ones will, especially if direct or even indirect laryngoscopy is very difficult or for any reason impossible. I have in mind two patients with ankylosis of the mandible, as a part of a general joint infection due to tonsillitis, in whom it would be difficult or even impossible to examine the larynx except by transillumination. In fact, the ankylosis is so pronounced that it is difficult for them to masticate properly. While such cases are the exception to the rule, they do occur.

Schmidt,[7] however, has the following to say: "For laryngeal examinations, in which the transilluminator with a cover, as in frontal sinus examinations, is placed anteriorly on the larynx, I regard the method as without value. Besides, it is very difficult to place the mirror in the correct position in the dark pharynx. It is true that a transilluminated larynx gives a beautiful picture, but I have not obtained any practical results from the method."

Roth[11] does not favor transillumination of the larynx, as he believes the test is not practical.

Dr. H. Arrowsmith writes as follows in a letter: "Transillumination of the larynx was done, to a very limited extent, twenty-odd years ago. I tried it out casually at that time, but with others discarded the procedure as having no practical value."

The normal larynx shows shadows cast by the thicker portions, such as the true and false cords and the cricoid cartilage. The thyroid cartilage offers, of course, the greatest obstruction

to the rays of light. The rings of the trachea cast very definite shadows.

The ventricle of the larynx, the cricothyroid membrane, and the membrane extending between each two adjacent tracheal rings admit more light than the thicker structures, hence, at any of these points inflammatory thickenings, benign or malignant growths, will show most distinctly on account of the shadows which they cast. The same may be said of the membrane extending from the lower border of the cricoid to the first ring of the trachea.

During the past fifteen months I have had under observation a papilloma of the larynx in a man, aged forty-one years. This growth has returned twice following removal by suspension laryngoscopy. The papilloma originally sprang from the left ventricle, but later seemed to be attached to the inferior surface of the false cord, too. This casts a distinct shadow by transillumination, during suspension laryngoscopy, and shows well the inflammatory thickening at the point of attachment. While the pathologist reported a suspicion of malignancy from the second papilloma removed, I do not believe it is malignant, but the shadow cast is due purely to the thickened tissue.

Cases examined with the bronchoscope or esophagoscope for a foreign body or a stricture, it is needless to say, show only the normal shadows. Imperatori states in a letter: "I have noted the light passing through the cricothyroid membrane while suspending the patient and with the long lighting attachment within the larynx." Since I have not had an opportunity to use this in a single case of malignancy, I cannot speak from experience of its value here.

J. Solis-Cohen in a personal communication writes as follows: "I have tried transillumination of the larynx and trachea, but got very little satisfaction from it. The parts looked like translucent candy, and it was not possible to outline growths and structure, so I gave it up. I do not think I ever wrote upon the subject."

Schech[5] makes the following interesting statement: "The transillumination of the larynx, first practiced by Czermak, and later being perfected by Voltolini, has no diagnostic value."

Dr. Frank E. Miller writes as follows: "I have had at least fifteen years' experience with transillumination of the larynx and trachea. In each I find it especially helpful, where there are gouty deposits and tuberculous combinations. It has proved extremely satisfactory and has been particularly helpful in diagnosing cancer cases, by exclusive processes. At the present time I have three cases of gouty deposits of the thyroid and arytenoid where the above had not been employed, causing improper recognition and tracheotomy. In a number of cases where this has shown simply a shadow overlying the thyroid gland, the penumbra has resulted in serious errors of diagnosis."

Frankel[8] has referred to transillumination of the larynx in Simon's handbook of laryngology, but mentions this very briefly.

Dr. Norval H. Pierce states: "I have had some experience with transillumination of the larynx and trachea, and now have a paper in preparation on the subject. This procedure has been used before, as a paper was read on the subject before the Belgian Laryngological Association."

A. L. Benedict[9] has suggested the use of transillumination of the upper respiratory tract.

Wm. Cheatham, in reply to my letter of inquiry, states: "A few years ago I became interested in laryngeal transillumination and used de Zeng's frontal sinus transilluminator. I found the study very interesting."

Dr. Lee M. Hurd writes as follows: "Four years ago, while observing a bronchoscopic tube being passed in a darkened room, I noticed the translucency of the light on the neck and chest wall as the tube was passed, and later reversed the idea of light outside, using mirror to examine larynx and trachea. I have used the method for observing the contrast and to outline growths in the larynx. Lennox Brown used a similar method thirty years ago."

To quote further from Freudenthal:[1] "Now, to get a view of the interior of the larynx, we apply this lamp to the external surface of the neck, either at the pomum Adami or near the cricoid cartilage, and introduce the heated laryngoscopic mirror into the dark pharynx, and thus we obtain a splendid view of it. But this view is quite different from that ordinarily

seen. The first thing that strikes the observer is the disappearance of different colors. The whole larynx shows a reddish tint of varying intensity. When we place the lamp at the level of the incisura thyroidea, then the vocal bands and all parts above them appear of a beautiful red color (of course the epiglottis is dark). But when we place the lamp near the cricoid cartilage, then we get a better survey of the whole subglottic region down to the bifurcation of the trachea, and this view is often more satisfactory than that obtained by the common method. But it must be observed that we have to become accustomed to this peculiar view of the larynx, for, it may be said, we see mostly in the negative. What, in ordinary illumination, impressed us as a thickening or an enlargement of a solid mass, in transillumination strikes us as a dark object. The rays of light cannot penetrate, and we infer that a solid mass, or a mass subject to the same optical laws, must intervene. We will, therefore, be able to define more precisely the contour of a tumor, because we see how the mass differentiates itself sharply by its dark outlines from the other parts in view. Only a few days ago I saw a patient of Dr. L. Weiss, who had a tumor on the posterior wall of the larynx, extending between the two cartilages from the left arytenoid downward. We can see the circumference of it by means of transillumination better than by the ordinary method. I will have the pleasure of showing you the patient, and will be very glad to have the benefit of your opinion upon the case."

"Furthermore, we will be enabled to differentiate, in a certain case, between a solid tumor and a cyst, and this point seems to me to be of sufficient importance to give the method a fair trial. A few weeks ago I operated on a man who, a few minutes afterward, happened to be seen by Dr. Hoch, of Philadelphia. The patient had a tumor, the greatest part of which was below the glottis. It was attached to the right vocal band at the front angle, and extended over a little to the left vocal band. By means of transillumination I could easily determine that it was a solid tumor, not a cyst, and could gauge the exact size of it. It is true that you could have excluded a cyst in this case even without transillumination, but there are cases where this is not so easily done, and for these I think transillumination is also of value."

"In this man, by the way, we could see, the day after the operation, that the edematous vocal band was much more translucent than before the edema had set in, and this brings us to the other class of cases, where we have a greater penetration of light than normally. This, however, can only be seen where loss of substance has taken place, as in ulcerative processes or in other pathologic conditions which admit a greater penetration of light. When Gottstein[10] says, 'the reddened vocal band seems just as translucent as the normal,' he is correct; but you cannot draw from that a conclusion as to the usefulness of this method generally. Certainly we cannot recognize these changes, but there are others which we can see so much the easier. So, for example, an edematous vocal band is differently translucent than an infiltrated one. The first is of a strong, red light—i. e., much redder than the normal—the last is much darker. This is a differentiation which is interesting enough to us."

While I have used transillumination in a few cases, my experience is too limited to warrant many positive conclusions, so I will, therefore, submit this test or method of examination for your approval in this "first communication," but I shall report more in detail later.

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XXXI.

THE RELATIONS OF THE SPHENOID SINUS TO THE SEMILUNAR (GASSERIAN) GANGLION AND THEIR POSSIBLE CLINICAL IMPORTANCE.*

By Greenfield Sluder, M. D.,

St. Louis.

The semilunar ganglion is at present thought of as so far removed from the sphenoidal sinus that they have not been associated in the minds of the anatomists or clinicians. The internal carotid artery usually rises on the lateral aspect of the body of the sphenoid, and the semilunar ganglion is usually lateral and posterior to the ascending artery. This, however, is not always true. The position of the artery seems to have a large part in determining these relations.

I present drawings of two specimens showing an intimate association of the sphenoid sinus to the semilunar ganglion, or parts of it. Figure 1 shows dissection of the cavernous sinus, semilunar ganglion and sphenoidal fissure, viewed from above. The sphenoid sinus, lying beenath these structures, is shown in dotted line. The thickness of bone separating the cavity of the sphenoid from these structures is egg shell thin. The internal two-thirds of the ganglion are exposed to the uppermost part of the sphenoid cell, and the external third is exposed at a little greater depth as the nerve tissues approach the foramen ovale. The mandibular nerve in the foramen ovale is exposed for ten millimeters to an egg shell bone separation from the sphenoid cell.

In the sphenoidal fissure the oculomotor, abducens and ophthalmic are exposed to an egg shell separation from the sphenoid cell. The trochlearis alone is not in this contact, because it lies on top of the oculomotor.

Figure 2 shows a sagittal section through the oculomotor

*Read before the meeting of the American Laryngological Association, May, 1916.

nerve within the cavernous sinus. The specimen is viewed from within outward. In this specimen the ganglion is exposed on its anterior limit for ten millimeters to an egg shell thin bone separation from the sphenoid sinus at the origin of the ophthalmic and maxillary divisions. The sphenoid cell measured 1.50 centimeters sagittal, 4.50 centimeters vertical, 5.50 centimeters transversely.

In 1912[1] I proved the permeability of the sphenoid sinus wall to small amounts of cocain. Following this observation, with its lesson, into these anatomical associations seems to me to offer an explanation of the herpes which develops in the wake of sphenoidal infections, or which arise in some patients from ordinary coryzas (irritation of the sensory ganglion cells being necessary for the development of herpes), and to explain why semilunar ganglion neuralgias and tic douloureaux of sphenoidal origin[2] sometimes recover as a more or less acute or subacute lesion, and at other times requires a ganglion removal or a posterior root section—i. e., sometimes the sphenoid lesion can be controlled and at other times it cannot.

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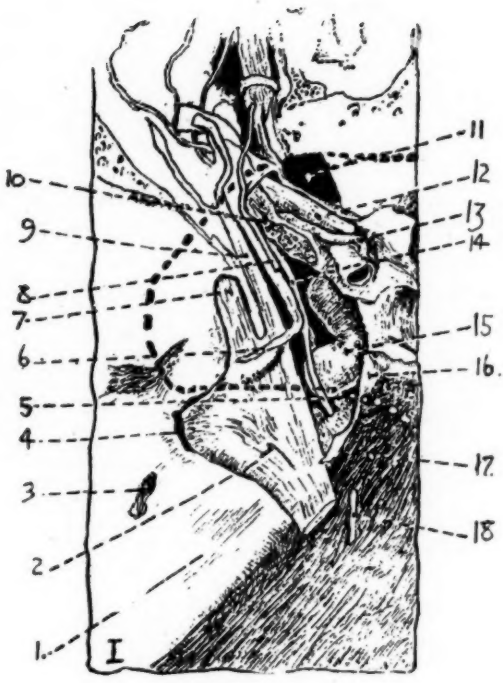

FIGURE 1.

Top view of left sphenoidal district, dissected. 1. Petrous part of temporal bone. 2. Semilunar (Gasserian) ganglion. 3. Foramen spinosum. 4. Foramen ovale with mandibular nerve in it. Inside the sphenoid cell the foramen ovale containing the mandibular nerve marked as a canal five millimeters long, separated in a semicircular exposure from the sphenoid cell by bone one millimeter thick. 5. Abducent nerve. 6. Oculomotor nerve. 7. Maxillary nerve entering foramen rotundum. 8. Trochlear nerve. 9. Ophthalmic nerve. 10. Inner limit of sphenoidal fissure. 11. Opening dissected into sphenoidal sinus. 12. Optic nerve. 13. Ophthalmic artery. 14. Abducent nerve. 15. Internal carotid artery. 16. Posterior clinoid process. 17. Clivus of Blumenbach. 18. Abducent nerve. Dotted line shows outline of sphenoidal sinus. It is separated from the overlying structures by a wall of bone, egg-shell thin, including those in the sphenoidal fissure. The inner two-thirds of the semilunar ganglion for almost one-half its length are also included in this close associati.on.

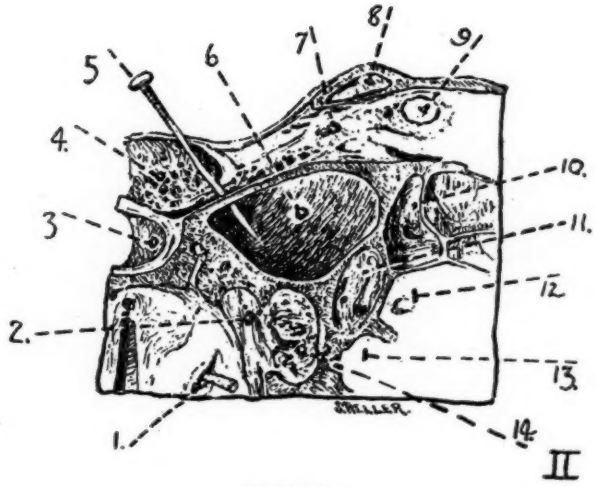

FIGURE 2.

Shows a sagittal section of left side through the ophthalmic nerve. 1. Eustachian tube with bristle in its lumen. 2. Tensor palati muscle. 3. Part of internal carotid artery. 4. Semilunar ganglion in Meckel's cave. 5. Pin passed through wall of sphenoid. 6. Sphenoidal sinus. 7. Ophthalmic nerve. 8. Lesser wing of sphenoid. 9. Optic nerve. 10. Postethmoidal cell. 11. Pterygomaxillary fossa. 12. Opening dissected into maxillary antrum. 13. Wall of nose. 14. Internal pterygoid muscle.

XXXII.

IS A MODIFIED RADICAL, OR HEATH OPERATION, A JUSTIFIABLE PROCEDURE?

By A. Spencer Kaufman, M. D.,

Philadelphia.

The modified radical, meatotympanic, or Heath operation, like many other surgical procedures advocated, is very much abused, both in its application and by its critics. Many times the one who recommends these methods is carried away by his own enthusiasm, and advises too free use of his operation, and when others fail to get results, there is general condemnation. Then, again, the users often fail to follow indications and to properly select their cases, and again the operation falls into disuse, regardless of the good there may be in it. I believe the Heath operation suffers for the first reason.

The modified radical mastoid operation was first advised by Körner in 1899, and later by Heath, Bryant and Ballenger. Jansen also claims to have described this operation in 1893.

Briefly stated, the operation consists in doing a simple mastoid or Schwartze operation, and the removing of the posterior bony wall of the canal to within a quarter of an inch of the annulus tympanicus, dividing the cartilaginous canal into flaps, and closing the posterior wound entirely. The contents of the middle ear are not disturbed in any way, and all postoperative treatment is carried out through the external auditory meatus. Every bit of mucous membrane must be removed from the antrum. In other words, you stop just short of doing a complete Schwartze-Stacke operation.

It has for its object the cure of suppuration, the repair of the drum membrane, and the restoration of hearing.

It has been asserted by some operators that a case requiring a radical operation will not get well with a Heath; and a case that does get well with a Heath would have made equally good

recovery with a simple; and this is true to a certain extent. But there is a certain class of cases to which this operation is so admirably adapted that I believe it has a real place in otitic surgery—not to supplant either the radical or the simple, but to take care of a group of cases in which it is indicated.

In a large measure, however, it can be used to advantage in a great many cases in which we have heretofore felt that an ossiculectomy would serve.

The cases of chronic suppurative otitis media which actually indicate the employment of intratympanic surgery alone are exceedingly rare; indeed, I had despaired of finding such a case until the past few days, when a case turned up with the qualifications for an ossiculectomy. This operation will answer in that very limited class of cases in which the suppuration is limited entirely to the tympanic cavity, the discharge being caused by granulations and necrosis in the cavity and of the ossicles. Here, as in a constantly increasing number of conditions, the X-ray plays an important part. Given a case of chronic suppuration of the middle ear, the X-ray examination showing the presence of cells in the mastoid process not filled with pus, not yielding to local treatment, the operation for removal of ossicles is indicated; and I submit an X-ray plate taken by Dr. Willis F. Manges, revealing just such a condition, and another plate, for comparison, showing an eburnated mastoid, in which we would expect to find an antrum necrosis, and in which the removal of ossicles would be of little, if any, service.

In cleaning out the tympanic cavity in a radical mastoid operation the malleus and incus are, in the majority of instances, found either whole or with very little evidence of necrosis. This is true, even though the otorrhea has continued over a long period, and the examination through the canal shows destruction of the entire tympanic membrane, and the ossicles are not seen in the tympanic cavity proper. This fact leads me to believe in the futility of an ossiculectomy, and the possibility of the repair of the drum membrane and the restoration of hearing when the ossicles are in almost normal position and only a part of the tympanic membrane destroyed.

In cases of chronic suppuration, with the destruction of the greater part or all of the membrane, and the ossicles missing,

or drawn up into the attic, and a roentgenograph showing either necrosis (pus) or a dense mastoid, but one operation is indicated, and that is the radical, or Schwartze-Stacke. As one of the objects of the Heath operation is the regeneration of the drum membrane, we certainly could not expect to have that result brought about when the ossicles have been drawn into the attic and could not again be gotten into normal position. Indeed, one would feel that if the membrane did reform under these circumstances, it would not act upon the ossicular chain, and impair rather than improve the hearing, thereby defeating the efforts for an improvement in function; and if the drum is not restored, we have only avoided the danger of injuring the facial nerve, and face the possibility of another operation.

For cases of acute mastoiditis not yielding to nonsurgical measures, the simple mastoid operation is unquestionably the one of choice. As the early and free incision of the tympanic membrane prevents all but a very small percentage of acute otitis cases from going to the point where a simple Schwartze operation is necessary, so in turn the simple operation, done early enough, will avoid the necessity for either a Heath or a radical operation.

If it is not intended to supplant either the simple or radical operation, where, then, are we justified in using it? There is a certain number of acute cases with which we come in contact that have gone too far to yield to a simple operation, and, on the other hand, do not demand a radical; or cases which would yield to a simple operation only after an abnormally long period of drainage and dressing. Prolonging the period of convalescence in this way frequently interferes with a patient attending to his usual work, on account of his wearing the bandage when he is physically fit to earn his livelihood. During the performance of a simple mastoid operation the bony canal is occasionally found to be very necrotic. To remove all of the necrotic bone would subject the patient to the danger of a collapsed canal, and to perform a radical operation under these circumstances would result in loss of hearing; whereas, if the tympanic ring is left intact and the contents of the middle ear cavity undisturbed, the result on hearing will be the same as in a simple operation. Then we find

chronic cases with the disease confined to the antrum and mastoid proper, and the ossicles in place and a goodly portion of the membrane remaining. The membrane, in these cases, after a Heath operation, will reform, and the hearing be much improved. The typical case for a Heath operation is one of this type. The patient complains of a discharge having continued for some period, and examination reveals a large perforation of the tympanic membrane and the ossicles plainly visible. The X-ray will show either necrosis or eburnation of the mastoid, with necrosis of the antrum.

To illustrate, I wish to report a case typical in its indications and consequently satisfactory in its results.

Mr. C. S., aged about forty years, in September, 1912, felt a sudden fullness in his right ear, examination of which, the following day, showed a very red and bulging membrane. This was incised and pus evacuated from the middle ear. Three weeks later he had a similar occurrence in the left ear. The discharge from both ears continued and resisted all efforts to control it, and in the meantime he was becoming progressively deaf. I advised a simple mastoid operation, which was refused, and he went to another physician. In February, 1913, he came back to me, the discharge still continuing, and a destruction of the lower third of the membrane on both sides. His hearing at this time was absolutely nil, and it was necessary to write anything one wished to communicate to him. A Heath operation was advised and consented to. His physical condition not being good, the two sides were operated upon a week apart, in February, 1913. The patient went on to an uninterrupted convalescence, the drums regenerated, and his hearing improved up to the point where it is now, and he has no difficulty in getting along with ordinary conversation. There was a small area in each mastoid where dermatization was delayed for some time, but this did not interfere with his hearing or middle ear.

CONCLUSIONS.

It is a justifiable operation in—

First—Chronic mastoiditis where there is only partial destruction of the tympanic membrane and the ossicles are in position.

Second—In cases of acute mastoiditis with an unusual amount of destruction of the tympanic membrane and loss of hearing.

Third—In cases of acute mastoiditis with extensive necrosis of the bony portion of the external auditory canal.

It should never be attempted where there are cholesteatomatous masses found in the antrum, which indicate the radical operation; and it should never be done until all nonsurgical methods, including vaccines, have been tried.

XXXIII.

A CASE OF ACUTE MASTOIDITIS WITH AN ENORMOUS LEUCOCYTE COUNT—BOTH MASTOIDS OPENED—RECOVERY.

By George F. Keiper, M. D.,

La Fayette, Ind.

On January 4, 1916, through the courtesy of Dr. E. T. Mitchell, of Romney, Indiana, I saw at my office Alva B. S., aged four years, of Clarks Hill, Indiana. The left mastoid was the seat of acute inflammation, with suspicious symptoms in the right mastoid.

Four weeks before, he had an attack of tonsillitis, which eventuated in a case of purulent bilateral otitis media. Tenderness of both mastoids was rather marked. Under the ordinary treatment the inflammation in the right mastoid seemed to clear, until at the time of his visit to me, as above, it did not seem at all tender. But not so with the left mastoid. It continued to grow worse, and hence he was brought by his doctor to me. The left ear stood out from the head markedly. Tenderness over the mastoid was exquisite. The posterior superior wall of the external auditory canal sagged markedly, hiding the upper portion of the membrana tympani. The drum was perforated irregularly in the posterior inferior quadrant. From the perforation pus issued under pressure. A smear of it showed a mixed infection with a large number of streptococci present.

The right mastoid did not seem tender at all, neither was there any sagging of the external auditory canal present in the right ear. Few streptococci were found in the discharge of pus from the ear. The membrana tympani was ruptured irregularly in the posterior inferior quadrant.

The boy was sent to St. Elizabeth Hospital for blood counts, X-ray plates, and urinary analysis. The urine was normal.

The blood count was as follows: Hemoglobin, 90 per cent; leucocytes, 60,090; polymorphonuclears, 91 per cent; small lymphocytes, 8 per cent; large lymphocytes, 1 per cent; mast cells, none; eosinophiles, none.

Astonished at this enormous leucocyte count, I had another one made the same day, which verified in all respects the first one. In fact, it is the largest leucocyte count that I have ever encountered in mastoiditis. Search of the literature fails to reveal to me a parallel in this disease.

His temperature on admission to the hospital at 4 p. m. was 100.2°, and his pulse was 134.

I advised opening the left mastoid as soon as possible. X-ray plates showed it cloudy; the right one seemed normal. This consent being given, we performed a simple mastoid operation the next morning. The cortex was thin, and pus was soon reached directly under it. All diseased bone was chiseled and curetted out clean. The wound was sewed up above, packed lightly and bandaged, and the patient returned to bed.

On January 6th the leucocyte count showed a decided drop to 16,500 per cubic millimeter. The polymorphonuclears dropped to 80 per cent, the small lymphocytes increased to 18 per cent, and the large ones to 3 per cent. By January 8th the leucocyte count had further dropped to 11,000 cubic millimeter. The polymorphonuclears had dropped to 72 per cent, the small lymphocytes had increased to 22 per cent, and the large ones to 6 per cent.

After the operation on January 5th the temperature at two o'clock was 99°, the pulse 144. By seven o'clock the temperature was 102° and the pulse 162. At eleven o'clock that night the temperature was 100°, but the pulse remained high, 160.

The temperature dropped on the 6th to 98°, with a pulse of 130. On the 7th it had dropped to 97°, with a pulse of 118. On the 8th the highest temperature recorded was 99.2°, with a pulse of 124. On the 9th it was 99°, with a pulse of 120. On the 10th the highest temperature was 98°, and the pulse was 116. The 11th, 12th, 13th and 14th showed like records. But the leucocyte count had increased, as shown by the chart of the blood count submitted herewith. It has been my

own observation that when the pulse runs high out of ratio to the temperature, further trouble is brewing, and frequent blood counts are indicated to warn us of further infection.

On January 5th the temperature was 100.8° by axilla, and the pulse was 148. At five o'clock it was 101.4°, and the pulse 140, and so it continued until the 16th, when a marked swelling appeared behind the right ear over the mastoid process, with increased tenderness and bulging of the posterior wall of the external auditory meatus. The blood count showed 15,300 leucocytes per cubic millimeter, and the percentage of polymorphonuclears was 80. The pulse was 130 and the temperature was 100°. I advised opening the right mastoid, which was accomplished by a simple operation at eleven o'clock the same morning.

The first incision through the skin brought pus in abundance. In uncovering the mastoid process we found a perforation in the bone about five millimeters in diameter. With chisel, curette and burr we cleaned it out. In doing so we found the tegmen eroded and necrotic, and in removing this we uncovered the dura mater, which seemed normal in all respects. The wound was dressed as in the first instance and the patient was returned to bed. By two o'clock the temperature had dropped to 98°, with a pulse of 130.

As will be noted by a study of the accompanying temperature and pulse chart, the former ranged rather high until January 21st, when the temperature rose to 104.6°, at 7:30 p. m. At this time tincture of aconite in drop doses every hour was given until the temperature was reduced. No symptoms of meningitis or lateral sinus obstruction at any time were apparent. By January 22nd the temperature had fallen to normal, with a pulse corresponding to the temperature. Barring a few exacerbations, controlled by tincture of aconite, it continued to remain normal, with a normal pulse, until dismissed from the hospital, February 8th. The blood count in the meantime had become normal.

In addition to the aconite the internal treatment consisted in giving three grains of hexamethylemin tetramin every four hours during his waking hours during his stay in the hospital. Paregoric in ten drop doses was used to control restlessness, which was quite marked at times, especially at night. Quinin

bisulphate in oleic acid was occasionally rubbed into the abdomen for the control of the temperature, though it was not as efficient as the tincture of aconite. The bowels were kept well open at all times, and high rectal injections were used to assist the medicinal treatment. Twice during the progress of the treatment the kidneys refused to secrete normally. Basham's mixture (liquor ammoniæ acetatis, fl. oz. vj; acid acetic, oz. iij; tinct. ferri chlor., fl. oz. v.; alcoholis, oz. ij; syrup, fl. oz. iv; aquæ, fl. oz. iv) was given with good effect. Hot stupes were also used over the kidneys. On the 17th and 20th a mixed stock vaccine was given hypodermically. Its value was very doubtful.

The most unusual feature of this case was the enormous leucocyte count of over 60,000 white cells per cubic millimeter. For a four-year-old boy, with comparatively small mastoid antra, it seems almost inconceivable to say the least. But the repeated blood counts, made the same day, leave no doubt as to the correctness of the observation.

As already intimated, the study of the temperature and pulse chart is fraught with interest.

Another interesting fact is that with so grave an infection the patient recovered, showing the great resistance possessed by this lad.

Were I to do this work over again, I would most certainly open both mastoids at the same time, in spite of negative X-ray diagnosis.

THE LEUCOCYTE COUNTS:

	Jan. 4	Jan. 6	Jan. 8	Jan. 10	Jan. 11	Jan. 12	Jan. 13	Jan. 16	Jan. 17
Leucocytes	60,090	16,500	11,000	12,400	11,600	15,200	15,600	15,300	15,000
Polymorphonuclears	91%	80%	72%	76%	84%	84%	84%	80%	77%
Small lymphocytes	8%	18%	22%	19%	12%	9%	11%	17%	19%
Large lymphocytes	1%	3%	6%	5%	4%	7%	5%	3%	4%
Mast cells	0%	0%	0%	0%	0%	0%	0%	0%	0%
Eosinophiles	0%	0%	0%	0%	0%	0%	0%	0%	0%

XXXIV.

AN OBSCURE CASE OF SIGMOIDAL SINUS THROMBOSIS.

By T. H. Odeneal, M. D.,

Beverly, Mass.

Patient, seen day after admission to the hospital, complained of pain in the mastoid region, not severe, tender about the tip; ear discharged a thick creamy pus. Temperature normal in morning, elevated to 99° in the evening. Patient was a thick set, robust German, and did not understand why I wished to operate, as he felt in such perfect health. Duration of symptoms one week.

Believing I had a simple mastoiditis alone to cope with, the mastoid was opened and a fistula found leading from beneath the cortex to the sigmoidal sinus, the bony covering of the sinus being entirely absent and the sinus found filled with a clot from the knee to the bulb. The jugular was ligated and the clot turned out, the patient making an uneventful recovery.

This case is interesting because of the lack of diagnostic symptoms pointing to a sinus infection.

I am almost led to believe that the thrombosis was an earlier affection and the mastoiditis merely a recrudescence of a previous one, which would account for the lack of any more definite symptoms.

Another case I have recently operated upon was seen with a large swelling behind the ear, reaching from the tip of the mastoid into the temporal fossa, which fluctuated on palpation. The temperature was normal, occasionally reaching 99° in the evening, and there was absolutely no pain or tenderness whatever.

A diagnosis of postauricular abscess of unknown origin was made, without an involvement of the mastoid. About two ounces of a creamy pus escaped on making an incision in

the fluctuating mass. About two centimeters from the tip two small fistulæ were discovered which led on probing deep into the mastoid cells backward toward the sinus. On following up these two fistulæ with the chisel they were found to end at the membranous wall of the sinus, the bony covering having disappeared for a distance of two centimeters or so.

No granulations being present on the sinus, and the pulsation apparently normal, the sinus was not opened. The remainder of the mastoid was very much sclerosed. Patient is now making a rapid recovery, temperature remaining normal since operation, six weeks ago.

These two cases are very good examples of how utterly impossible it is to judge of the extent of a mastoiditis from a symptomatic standpoint, and how necessary it is to be on our guard constantly.

XXXV.

COMPLICATED GRADENIGO'S SYNDROME—OPERATION—RECOVERY.

By Adair-Dighton, M. D.,

Liverpool.

The following case seems, on account of the extreme diversity of the symptoms, to be worthy of report:

John B., aged sixteen years. Admitted to the Eye and Ear Infirmary on August 9, 1916, with a history that he had been taken suddenly ill four days previously.

On examination, patient was obviously very bad. Was reported by own doctor to have had a rigor; was semiconscious; complained of intense frontal pain, tenderness over left mastoid, profuse discharge from left ear. Temperature, 104°; pulse, 104; respirations, 28.

Dr. Edgar Stevenson examined his eyes, but reported both discs normal. Marked nystagmus of left eye to right side. Left ear not totally deaf.

On following morning he was moved to the operating theatre, and when on the table it was noticed that he had developed a marked strabismus in the right eye to the left, which was easily proved to be due to a complete paralysis of the right external rectus muscle. This had not been present on the previous evening. The right ear was normal, but there was a history of a "one-time" suppuration in it. Under chloroform anesthesia I operated and performed a very complete radical mastoid. The antrum was full of semifluid pus under pressure, and, as might be expected from symptoms, there was a distinct fistula in the external horizontal canal. Dura and sinus were purposely exposed and found healthy.

On following day the temperature had dropped to normal, where it remained throughout convalescence. Pulse, 98. Nystagmus and squint, no change.

The convalescence was normal, the nystagmus ceasing three weeks after operation, and the strabismus gradually clearing until on the 16th of September patient was discharged with the external rectus acting perfectly and a dry left ear. He had no refractive error.

Now this case is of interest:

1. On account of the alarming character and diversity of the symptoms.

2. The paralysis of the opposite external rectus.

3. The sudden cessation, after operation, of the alarming symptoms.

First, the symptoms of the typical Gradenigo syndrome have been so well described in the ANNALS by Hays, Perkins, Myers and others, that no addition of mine need be added, but throughout the whole of the literature on the subject I can find no mention of a case in which the abducens paralysis affected the opposite eye, and am at a loss to account for it in any way except the possibility of a reflex action lighting up an old-standing focus of disease in the right ear, and so causing a neuritis of the sixth nerve on the right side. Even with this as a possible explanation, I cannot see that it accounts for the absolute lack of symptoms in that ear, as there was never any sign of discharge. I have thought of the possibility that the right eye was a squinting eye of old standing, but failed to get any history of it, and there was no error of refraction.

SOCIETY PROCEDINGS.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of January 12, 1917.

Paper: The Practical Application of Recent Work on the Internal Ear.*

By Isaac H. Jones, M. D.,

Philadelphia.

(By invitation.)

Paper: Vertigo: Its Causes and Methods of Diagnosis by Ear Tests†

By Lewis Fisher, M. D.,

Philadelphia.

(By invitation.)

DISCUSSION OF DR. JONES' PAPER.

Dr. B. Alex. Randall (Philadelphia) said: Supporting the claims of his colleagues, he would add little to what they had presented, but rather enforce some points perhaps too lightly touched upon; for he adopted the maxim of Michael Angelo: "Perfection is made of trifles; and perfection is no trifle." The chair which Dr. Jones has designed and illustrated is a considerable improvement upon Bárány's, still more upon the substitute shown in some of the pictures, since it places and holds the head in the exact plane desired, as near as possible to the axis of rotation. While nystagmus may be well evoked with approximate arrest of the turning, it is to be noted that for past-pointing the chair is always locked—best by a pedal. Poor appreciation of the exact planes of the

*See page 436. †See page 511.

semicircular canals has given rise to much faulty testing; the external canal, which many of us incline to call the horizontal, is thirty degrees from the horizontal, as shown; while the vertical canals are each some forty-five degrees away from either the sagittal or the frontal plane, by which some would name them—facts of fundamental importance. Then, just as we must abandon the misleading names like "crista acoustica" and "macula acoustica" for structures devoid of acoustic function, so must we cease to speak of the "static labyrinth" when dealing with the canal apparatus concerned in the perceiving and analyzing of motion. This is kinetic, not static.

The primordial aural organs, with their otoliths, are mainly static; although they may show not only the direction of gravitation, but somewhat appreciate motion and their hair cells recognize sonorous as well as other vibrations. Only in the more mobile animals, as the fish, does a semicircular canal apparatus appear, with a nonotolithic end organ in the ampullæ capable of recognizing and analyzing endolymph flow. The sacs of the vestibule have even in man their otolith organs, and it is of interest to note how the two "maculæ" are set at right angles to each other the better to fulfill their static function.

Man, trying to improve upon nature, has copied his camera from the eye, and in some essentials his telephone from the ear; and the aviator, in designing a "stabilizer," can probably do no better than follow the suggestion of the semicircular canals in their three planes at right angles to each other. For we may take it as the essence of all these phenomena—separate and independent, as Dr. Jones has told you the nystagmic are from the vertiginous symptoms—all are efforts, though perhaps perverted, to stabilize the sensorium. The patient feels as if failing in one direction, overcorrects, and so falls to the opposite side. He points to an object of known position; but while his hand is moving away and back, he changes his aim in exact proportion and direction as he subconsciously conceives his body to have turned from its primary position. Thus he gives a quantitative as well as qualitative indication of the stimulus of his kinetic apparatus. This is exactly the use which Graefe made of this test, using it to demonstrate the extra innervation given to paretic eye muscle.

This quantitative value requires that the tests be made with minute accuracy, unmodified by extraneous and perhaps calculated corrections; and shall be as rigidly recorded. To this end the charting which shall place this mass of facts clearly, almost graphically, before the examiner so that at a glance he can see all the essentials and note any flaws or perversions of the findings, is of prime importance to the securing of all the facts and to the analyzing of their meaning. Yet we have seen men try to carry in their heads the results of their inadequate testing, summing up as they went along to a futile conclusion; and then ready to condemn the whole investigation as worth no more (in this and presumably every other case) than the primary observation that the patient walked like a cerebellar ataxic. My colleague said: "Doctor, I never smiled once." It was really pitiable—rather a cause for tears. Only when well charted, often as the result of repeated or divided studies, is the case open to intelligent analysis, unless its showings have been immediately and consistently normal. The critical tracing of the paths involved is indeed neurologic, but only to be learned by these ear tests, and therefore neuro-otologic.

The great effort of our work has been to simplify and correlate and interpret: not by trying to state vertigo or past-pointing in relation to nystagmus, since nystagmus or even conjugate deviation of the eyes may be lacking or perverted; but to recognize and state that the stimulus is in absolute relation to the direction of endolymph flow. But this cannot always be determined with the "fistula test," hence its limited usefulness. It can be falsely simulated by galvanism, which can act on the nerve trunk or on the nuclei. Hence, our limited use of these tests and distrust of their showings. Enough that by reasonable use of the caloric and turning tests we can learn, often with extreme precision, facts otherwise unobtainable, especially as to subtentorial conditions, of utmost value to the otologist, the neurologist and the surgeon.

Dr. Charles A. Elsberg said he had heard so much that was interesting and instructive that he did not feel he could add much, if anything, to what had been said. He wished, however, to express his thanks to the gentlemen who had come this evening from Philadelphia and presented the results of their large experience.

He had been asked to say a few words on the subject from the surgical standpoint. Of course, from the standpoint of the practical surgeon, it is important to know what value these tests really have. Can they make the diagn for us? Can they make a differential diagnosis? When d fail? These are the questions which the surgeon must have answered—and they have arisen in a number of instances in which he had had these tests made for him.

Of course, before speaking of conclusions, it must be remembered that this work is still in its infancy, and that much more can be learned and will be learned in the future. Therefore, one should hesitate very much before saying anything derogatory to the tests or the results obtained by them.

The questions that have arisen in his mind in the course of the last few years regarding these tests are these:

1. Can we make the diagnosis by their means? Naturally, his remarks would have reference to the diagnosis of those conditions of interest to the general surgeon. His experience has been that, in the first place, they have been very valuable in differentiating between frontal and cerebellar tumors. Thus, for example, the differentiation between a right frontal tumor and a left cerebellar tumor is often difficult. Not infrequently, it has occurred to the most experienced surgeons that a case turned out to be one of cerebellar disease after an operation had been done in the frontal region. In two instances in the speaker's experience, the differentiation could be made by these tests.

In the second place, he had seen a number of children suffering from a nystagmus the cause of which could not be discovered by various specialists—neurologists and others. Finally, cerebellar disease was suspected. In several of these children, the otologist solved the question by showing that it was a congenital nystagmus which had no special significance.

2. What value have the tests for differential diagnosis? He had already spoken of its value in differentiating between frontal and cerebellar disease. He has not, however, obtained much information on the Bárány tests in cases where the diagnosis lay between internal hydrocephalus and cerebral disease with secondary internal hydrocephalus. In hydroceph-

alus the symptoms are often very like those of bilateral or even unilateral cerebellar disease, and in these patients no help was obtained from the tests. In one case the hydrocephalus had been much improved by puncture of the corpus callosum, but even after this the otologist was unable to state from his examination whether there was a primary infection of the cerebellum. It would be interesting to note from Dr. Jones and Dr. Fisher if they have been able to differentiate between cerebellar disease and internal hydrocephalus.

3. He has seen the test fail in disease of the posterior fossa where the symptoms pointed to the opposite side of the cerebellar lobe. As is well known, the tumor in one lobe of the cerebellum may press the other lobe against the petrous portion of the temporal bone and give cranial nerve and cerebellar symptoms on this latter side. In patients of this kind the Bárány test had shown an affection of this latter cerebellar lobe when the real lesion was on the other side. It may have been that the tests were incomplete or not thoroughly done. Perhaps in the hands of Dr. Jones and Dr. Fisher results would have been different. It would be valuable to note whether the failures were mistakes, or whether they were due to some inherent weakness of the tests.

All the foregoing remarks, however, are made in no sense of criticisms of the valuable tests which Bárány had described, which have much furthered our knowledge of vertigo and of the static function of the vestibular nerves.

Dr. Arnold Knapp: It is quite true that nystagmus is generally regarded by ophthalmologists as an associated symptom and of little diagnostic significance. To those, however, who have been fortunate enough to hear Dr. Jones' admirable demonstration and to see his remarkable moving pictures, nystagmus will have assumed a new meaning.

As for the practical value of the vestibular tests in nystagmus and in strabismus, though we have made at the Eye Hospital a routine examination of suitable cases, our experience is far from complete. In the form of nystagmus which is most familiar to us, the socalled amblyopic nystagmus which occurs in children with defective sight, albinism or retinitis pigmentosa, the vestibular reaction has been found unchanged. There is another group of cases of nystagmus in children

without any ocular signs of amblyopia who are suffering from congenital lues. In these cases Heine has found the spinal fluid to be under increased pressure. A case which recently came under the speaker's observation was that of a child of four years, who had suffered from right hemiplegia, then convulsions and nystagmus. After lumbar puncture the nystagmus and convulsions ceased.

In the cases of strabismus, those with the ordinary concomitant convergent strabismus, we have not been able to find any change in the vestibular reaction. This does not agree with Bartels, who found a disturbed reaction in nearly half of thirty patients. In two cases of nystagmus combined with convergent strabismus which we recently had the opportunity of examining at the Eye Hospital, the reaction to the vestibular tests was unaffected. Paralysis of the ocular muscles would seem to offer a field for this form of examination, and investigations along these lines have been made by the men in Vienna.

The vestibular tests offer a means of distinguishing between a supranuclear and a nuclear lesion, and if the reaction is normal the lesion must be supranuclear. Supranuclear lesions, however, are usually of associated movements, such as conjugate paralysis, conditions which are generally found in severe nervous disease and do not often visit the eye dispensaries.

This subject is surely a very interesting one, though to my mind it presents many difficulties; still, after listening to Dr. Jones' exposition, we should feel encouraged to persevere in our examinations.

Dr. Isidore Friesner said that in listening to these very interesting papers there would seem to be two topics for discussion. First, a consideration of the physiologic activity of the static labyrinth; and, second, the question which Dr. Knapp had just brought up—a consideration of the basis upon which conclusions from functional tests may be drawn.

With regard to the physiology of the static apparatus, he had so often and at such length discussed this question with his friends from Philadelphia, that it would be passed by at the present, except to say that it is not necessary for one to adhere to the cerebral or the cerebellar origin of the past-pointing in using the pointing test. The origin of this phenomenon

is not essential, or at all events not vital to our conclusions. Whether we believe past-pointing to be cerebral (the objective expression of the vertigo), intermediated by the cerebellum, or whether we hold that past-pointing is purely cerebellar, we use the pointing test in determining the disturbances of cerebellar function. From the point of view of the otologic surgeon, the mere presence of labyrinthine function is sufficient to warrant adequate conclusions. Of course, that is evidenced by the presence of hearing and the response to any one of the several stimuli which are applied to the static labyrinth. In making such tests we have all, time and again, seen decided differences in the responses from the two sides; or if we have tested the separate parts of the static labyrinth, we have seen decided differences in the responses from them; but because our conclusions were based entirely on the presence of function, these differences were disregarded.

From the neurootologic point of view, our conclusions are based largely on the intensity of the response and the promptness with which the stimulation arouses an impulse. This point of view is different from that of the otologic surgeon. For instance, with normal peripheral conditions, if we apply the same light separately to each eye, and find that the iris in one contracts promptly and fully, while that in the other contracts sluggishly and incompletely, do we not conclude that there is some disturbance of the reflex arc in the latter? Are we not equally justified in our conclusions if we find such differences after applying stimulation of equal intensity to both static labyrinths, even to the separate parts of each static labyrinth? For it is possible to stimulate calorically the horizontal and vertical canals separately.

Indeed, some recent tests of the vertical and horizontal canals separately, in cases of vertigo accompanying toxic conditions, accompanying purulent sinuitis—one case a compositor with lead poisoning—have indicated the possibility that there may be a retrovestibular neuritis analogous to a retrobulbar neuritis, in that certain fibers of the vestibularis are more susceptible than others; and, further, it would seem that the more susceptible fibers are those conveying the impulses from the vertical canals. The nerves have not been tested galvanically in these tests.

The diagnosis and localization of cerebellopontine angle tumors by the functional tests is, of course, comparatively easy. We, too, have observed in cerebellar pontine tumors the transmutation of the nystagmus impulses. Normally, the cold caloric test on the right side causes a nystagmus directed to the left; in several cases of cerebellar pontine tumor we have seen, following caloric stimulation, an oblique nystagmus or a vertical nystagmus upward. It would seem probable that the cross-pointing with cerebellopontine angle tumors, so well shown in Dr. Jones' "movies," is brought about by pressure on one or both lateral cerebellar hemispheres. Much of this work, as Dr. Jones said, is purely experimental.

Dr. Friesner concluded by saying that he would not wish to be misunderstood, particularly with regard to what he had said concerning retrovestibular neuritis. His statements were in no sense conclusions, but merely suggestions.

Dr. Joseph Byrne, after expressing his appreciation of Dr. Jones' wonderful presentation of the subject, said he himself had done a good deal of work upon the labyrinth. Some of Dr. Jones' statements were misleading, especially in regard to the precise mechanisms involved. Thus, in rotation tests, Dr. Jones said that the eyes are always drawn (long component of the nystagmus) in the direction of the movement of the endolymph, but he failed to state that during the actual rotation there is a primary nystagmus which, upon cessation or slowing down of the movement, gives place to the secondary nystagmus. It is this latter which is of opposite direction to that of the primary nystagmus that the observer studies. Dr. Jones said this secondary nystagmus is due to the continuation of the movement of the endolymph. This is true only in a sense, since Dr. Byrne had observed reversal of the nystagmus —e. g., from the primary to secondary form, with mere slowing down of the movement in rotations. It is well known, moreover, that the lumen of the membranous canals is of capillary dimension, and so precludes the possibility of such a thing as gross movement of the endolymph in rotations. What we are dealing with in rotation is not gross movement of the endolymph, but the most delicate alteration of conditions about the hair cells (slight bending), due to faint alterations of hydrostatic tension. A mechanism permitting of

gross movement of the endolymph would be quite useless for fine physiologic adaptations.

Dr. Byrne said that another thing to which he has always taken exception in Bárány's work is the assumption that, in the caloric tests, heat and cold produce their effects by causing movement of the endolymph within the membranous canals. Such movement, assuming that it does occur, implies displacement of the endolymph in opposite directions—e. g., currents of opposite direction arise, which, owing to the minute size of the canals, would, upon continuation of the irrigation, soon cease, owing to the equalization of temperature in the endolymph the whole way across the particular canal involved. This does not occur, as can be readily demonstrated; for the nystagmus persists in spite of prolonged irrigations. Numerous other objections might be urged against Bárány's view on this point, but Dr. Byrne contented himself with one experiment of his which seemed conclusive. It is well known that the labyrinthine receptors, especially those in the semicircular canals, are exceedingly sensitive to all kinds of stimuli—mechanical, electrical, thermic, and even photic. It is also well known that in any posture involving equilibration, tonus impulses are constantly streaming inward from the labyrinthine receptors, and that equilibration itself implies nice adjustments between the impulses coming in from the various labyrinthine receptors on either side of the body. In a word, in the labyrinths we have nicely adjusted balanced mechanisms. Assuming, as Dr. Byrne has done, that within certain limitations, heat exalts and cold depresses nerve function, as is well known to all physiologists, then prolonged cold irrigation should deaden the receptors affected and render them insusceptible to the effects of other forms of stimulation. This was put to the test. After irrigation of the left ear at 68° F. for four minutes the subject was immediately rotated from left to right about the vertical axis. On sudden interruption of the rotation the usual after-nystagmus was entirely missing, whilst the primary nystagmus to the left and against the watch persisted. This experiment, which seems conclusive, was put on record by Dr. Byrne as far back as 1910, but the work has been ignored or neglected.

There were statements in reference to vertigo made by Dr.

Fisher to which Dr. Byrne took exception. Vertigo is not a sensation in the ordinary sense, having a special receptor organ in the labyrinth, as Dr. Fisher states. This conception of vertigo was objected to by Ferrier over forty years ago. Orientation is an awareness or consciousness of our relations in space, and disorientation, of which vertigo is a form, is a disturbance in the sensory elements, etc., upon which this awareness is based. With this disturbance there goes, of course, a certain sense of insecurity, disordered equilibration, etc., which, from the subject's point of view, are the main features of disorientation. But all these do not make vertigo a sensation.

Dr. Byrne wished to call attention to the fact that the labyrinth has other and very important functions in the economy. He had long ago demonstrated its influence upon vasomotor function. Leonard Hill has shown that if a hutch rabbit be held upright it will, in from ten to twenty minutes, go into convulsions from cerebral anemia and die. Cats, dogs and wild rabbits, with their more efficient abdominal musculature, withstand the erect posture for much longer periods. Hill also found that if hutch rabbits be held submerged in water they will withstand the erect posture almost indefinitely without disastrous consequences. These facts are of intense interest where there is question of the erect posture and the influence of the labyrinth on tonus, whether of striped or unstriped muscle.

The labyrinth is also related to the vomiting mechanism. No vomiting center has as yet been demonstrated distinct from the respiratory center or that portion of it in direct relations with the effector mechanisms of expiration. It is probable that the otolithic apparatus (statoliths of Verworn) is in direct relation with the vomiting center. Every physician is familiar with the movements of the nares, lower jar, trachea and accessory muscles of respiration in impending death. These phenomena were matters of observation from time immemorial. Dr. Byrne said he had pointed out that they were all due to the progressively increasing demands made upon the great respiratory pump as an auxiliary to the heart and vasomotor system when the circulation begins to fail in the medullary centers. These phenomena are comparable to the dyspnea of asphyxiation and the general convulsions (both due

to increased hydrogen ion content of the blood) that close the scene.

Dr. Byrne pointed to the frequency with which vomiting or gastric regurgitation occurs at or near the point of death. The forced respiratory action and the vomiting are defensive mechanisms. They are the last reserves called out to stave off final dissolution. Disease, of recent years, has been defined in terms of reactions of the organism. In seasickness, which Dr. Byrne has studied extensively, it is probable that the vomiting and vertigo are protective reactions. In rotations and in seasickness Dr. Byrne has shown that one of the first effects upon the gastric mechanisms is closure of the pylorus. This is the first step in a train of phenomena which culminates in vomiting. This latter is a most powerful means of increasing the cardiac output, constricting the arterioles, and enhancing blood flow in general. In seasickness the medullary circulation is restored directly by the vomiting and indirectly by the vertigo which enforces recumbency.

Dr. Jones, in closing the discussion, said he thought Dr. Elsberg would find the following of help in the differential diagnosis between internal hydrocephalus and a lesion of the cerebellar substance: An internal hydrocephalus would as a rule show no nystagmus or past-pointing on stimulating the vertical canals of both ears, but stimulation of the horizontal canals of each ear would produce a normal nystagmus and a normal past-pointing. In a cerebellar lesion, however, no matter which canals are stimulated, we would expect a consistent absence of past-pointing of a certain extremity in a certain direction—for example, an absence of past-pointing of the right arm to the right. Further, if the cerebellar lesion did not involve the brain stem, we would expect a normal nystagmus on stimulation of all the canals.

Replying to Dr. Byrne, the following experiment would seem to prove conclusively that a movement of the endolymph must be accepted as the cause of stimuli from the internal ear: With the head back, douche the right ear with cold water; there occurs a horizontal nystagmus to the left, vertigo to the left, and past-pointing to the right. Without any further douching, incline the head forward; there immediately occurs a horizontal nystagmus to the right, vertigo to the right, and

past-pointing to the left. Simply by altering the position of the head we have produced diametrically opposite phenomena. This clinical fact can only be explained by an endolymph movement, first in one direction and then in the other.

DR. FISHER, replying to Dr. Elsberg's inquiry, said that these tests are valuable insofar as one is confident of his technic and is certain of the anatomic pathways which convey the results of stimulation. It is believed that when the normal ear is stimulated it will produce normal responses. When stimulation fails to produce normal reactions, it is always due to an interruption in these pathways. Such an interruption may be located in the ear itself, or in the pathways, or in the brain centers, and the ability to visualize these pathways, combined with a careful and painstaking technic, enables one to make a correct diagnosis.

As to Dr. Byrne's remarks about vertigo not being a conscious sensation, if you put an individual in a chair with the eyes closed and turn him to the right, he will say: "I am going to the right." Then, after you have turned him ten or fifteen times, and the movement of the lymph has caught up with that of the body, he will say: "I am standing still." As a matter of fact, he is being turned very rapidly, but because his lymph is moving as fast as his body, he is under the impression that he is not moving at all. Then suddenly stop the chair, and he will say: "I am going to the left." How is he to know all this unless it be a cerebral act?

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of February 9, 1917.

Remarkable Development of Mastoid Cells Following Fracture in Childhood.

DR. J. D. KERNAN: The patient was a young man, eighteen years of age. His family history had no bearing on his condition. He had had the usual diseases of childhood, and frequently suffered from colds in the head. Seven or eight years ago he fell four stories, but does not know where he was hit. He could not walk for three months, but does not think he was paralyzed or had any bones broken. Has been hit on the head a number of times. He has always been perfectly well, and has had no earache or trouble of any kind.

Two months ago, by chance, he noticed a swelling behind the left ear, which grew steadily larger, especially when he blew his nose. Before that he had always heard well; now, at times he does not hear so well, and then again hears perfectly. When he presses on the lump he does not hear so well in that ear. When he moves or walks he hears a crackling over the lump, somewhat like crackling in ears when swallowing. He thinks he was feverish once in the last two months. His appetite is good, bowels regular, and general health good.

X-ray pictures were presented for examination.

The examination of the ears gave rather negative results. The hearing of the upper and lower tone notes seemed to be normal. When the enlargement was pressed on there seemed to be a slight diminution in the hearing—a diminution of the air conduction and a lengthening of the bone conduction, but the changes were very slight.

Dr. Kernan said that three possibilities were to be considered as the cause. One, that the enlargement was merely a natural increase of the process of growth in the mastoid cells, such as is seen in the lower animals and especially in the anthropoid apes, where the mastoid cells extend into the surrounding bones; second, the possibility that when he was

injured the bone was broken in such a way as to give freer access than usual to the mastoid from the middle ear, and the blowing of his nose has caused pressure of the air into the mastoid and continued the process of cell formation; and third, that there may be some new growth, such as angioma or sarcoma, growing from the region of the middle ear and gradually boring its way through the bone.

DISCUSSION.

DR. LAW said that he had brought the plate down to find out what the members of the section thought about it. He himself did not know what to say about it. The picture on the plate certainly seems to be that of an enormous development of the mastoid cells. He did not think that it was a new growth, primarily, as it did not seem that a new growth would have eroded the bone with such a clear cut outline of cells; he did not know of any new growth that would do that. The pneumatic area, if one might so call it, seems to be in direct connection with the mastoid on that side, and extends to within half an inch of the opposite mastoid. One could conceive of a new growth superimposed on the mastoid, as accounting perhaps for the lump on the back of the head, but so far as he could make out from looking at many mastoids, this seems to be a mastoid structure. If it is anything else, he hoped someone would tell what it is.

DR. LEWALD thought the accumulation of air beneath the scalp the most difficult to explain. It might be called a pneumocephalocele, but that was only a name; and he had not the faintest idea what to say concerning the origin of the condition.

DR. LEDERMAN said he had never seen anything like it, and would like to know what Dr. Kernan proposed to do in the line of treatment. In his opinion, Dr. Kernan's explanation of the injury as being the cause of the condition seemed the most probable, from the fact that the young man can blow up and enlarge the mass, showing a direct communication from the middle ear. As he seems to be quite comfortable, it would seem questionable whether surgical interference would be justified, as it might do harm.

DR. HAYS said that the case reminded him of a bone sar-

coma, which is frequently seen in young children. In these cases there is a peculiar egg shell crackling which is distinctive and which can be felt in this case. From the X-ray plates and from the history of the case, it seemed to him that there was no definite anatomic connection with the mastoid bone. The X-ray showed the cell cavity extending over almost all the bones of the cranium. Of course, it is possible that these cells connect with the mastoid cells and the middle ear itself, so that in forcing air into the ear such air could be sent through these various cells.

Dr. Hays said that he had seen cases of sarcoma of the antrum in young children, in which bone would become very thin and would pit and crackle on the slightest pressure. Such sarcomata are not very malignant and do not form metastases very quickly. He said he felt that this was a case of pathologic change in the bones which connect with the mastoid cell.

Dr. Herzig asked whether the X-ray was taken of the left side, or of the right side, or both. To which Dr. Law replied that pictures were taken of both sides, and the mastoid cells were within one-half inch of the other side.

Dr. Haskin asked if anyone had any idea of what caused the crepitation.

Dr. Wilson, replying to Dr. Haskin, asked if it might not be the air in the scalp.

Dr. Haskin said that it felt as though there was a thin plate of bone that could be pressed down.

Dr. Wilson said that the crepitation disappeared when the air disappeared, and there was the distinct feeling of air under the scalp after blowing the nose.

Dr. Law said that with the examination made with the stereoscope there was no thin area as compared with the rest of the mastoid. In other words, the floor of this soft mass throws as dense a shadow as the rest of the area, so that if there is an increased thinness of the bone, it does not show in the plate.

Dr. Kernan said that as he understood the egg shell crackle in bone sarcoma, it is due to a thin plate of bone over the sarcoma, but he could not make out any bone over this soft tumor; it seems to lie directly under the skin without any overlying bone at all. What few cases of sarcoma of the bone he

had seen always showed the bone about the sarcoma in a state of rarefaction—thin, diseased looking bone. The bone in this case is extremely healthy looking except for the presence of the cells. He did not feel that he could accept the diagnosis of sarcoma, on account of the healthy appearance of the bone in the picture. As to the crackling, one sometimes sees the same thing in knee joints where there is inflammation. The synovial membrane being thickened will crepitate in the same way as this swelling when it is palpated; and there might be some inflammation present in this case, with thickening of the walls; or there might be, as the result of the old injury, a few loose bits of cartilaginous material which crepitate like a loose cartilage in the knee joint. He was inclined to consider the condition to be a result of the injury, which opened the way for undue extension of the mastoid cells.

A Case of Unilateral Aural Microtia With Unusual Features.

Dr. Wm. H. Haskin: The patient was a boy of eleven years, who had recently come to the clinic. Dr. Haskin said that the condition spoke for itself, and there was little to be said about it excepting that it was a case of congenital microtia with a very peculiar lack of development of the inferior maxilla. There was no zygomatic process, the bone ending abruptly at the malar process. There was some hearing on the defective side. With the Bárány apparatus in the good ear, the patient recognized the difference between a whistle and the loud ringing of the fork, and he could hear loud shouting; he recognized that it was a voice, though he could not detect words. Several X-ray pictures had been taken, and could be seen by any who were interested in looking at them. Rotation in the chair showed an apparent loss of function of the static labyrinth. Rotated to the left, there was a long nystagmus to the right, and vice versa.

Dr. Haskin said in all the years that he had been on the service he had not seen any cases of microtia until recently, when this case and another one had appeared. The other patient was a man with a deformed ear in the normal position, and his rotating test showed the same thing—when rotated to the affected side, he had a long nystagmus to the right; but

when rotated to the right, there was nystagmus to the left of only five seconds.

He realized that nothing could be done for this boy. There was no appearance of ossicles; from the stereoscopic plates there seemed to be a lack of development of the greater wing of the sphenoid. If there was any wing forming the floor of the middle fossa, it was very, very thin.

DR. PAGE said he did not think it was an operative case, as no case with one good ear is, and he did not think anything could be done for it. It was an extremely interesting condition. He did not understand why there should be the associated maldevelopment of the jaw. It is known that in cases of microtia there is usually a lack of development of the anterior wall of the canal, and of the anterior superior wall of the tympanum, with drum membrane and ossicles possibly lacking, but with the footplate of the stapes present, the internal wall of the tympanum present, and a practically good labyrinth present. Alexander gives as the reason for frequent failure in operations for microtia the attempt to enter along the line of what seems from palpation to be the line of the external auditory canal, as in this direction one encounters the jaw or the facial nerve. That is why Jaeger, in 1830, devised the operation of entering the mastoid antrum and working forward and letting the sound wave reach the stapes through the aditus. A point of diagnostic value made by Alexander is that where there is lack of development of the internal ear, there is almost always associated with it some facial paralysis. From the appearance of this case, he was inclined to believe that there was neither tympanum nor ossicles, but a stapes and internal ear.

DR. LEDERMAN said that a number of years ago he saw a bilateral case of this kind—i. e., a child with a similar deformity on both sides. This child could hear a loud voice and other loud noises The father and mother were both healthy and robust. When looking up the literature at that time, he had found that most men who had given attention to the subject agreed that operation offers no hope in these cases, as there usually existed a malformation of the middle ear. The late Dr. Hermann Knapp had spoken to that effect before the section a number of years ago.

DR. NORTON L. WILSON said that he had had a somewhat similar case, but there was not so much deformity of the jaw. The child had ossicles, but the closure was complete to the middle ear. The child had a little hearing, and he had endeavored to help the condition, but without any result. He was neither able to improve the hearing nor to keep the newly formed canal open.

DR. KERNAN said that the case was of great interest from the embryologic point of view, since the ossicles develop as part of the lower jaw from Meckel's cartilage. Therefore, in such a case as this where the caudal end of the lower jaw is undeveloped, one would expect that the incus and malleus would also be undeveloped. The stapes is developed from the proximal end of the hyoid arch, so that one would expect in this case to have the stapes present, and some hearing, whereas the conducting apparatus formed from Meckel's cartilage would be absent. The inner ear develops independently, by itself, and that might be perfect, while the middle and external ear are entirely absent.

DR. PAGE said that he was just about to ask what was the relation of the development of the jaw to the development of the tympanum and ossicles. Politzer states that (according to Gradenigo) the footplate develops in the wall of the labyrinthine capsule.

DR. KERNAN said there had been a great deal of discussion on this point, but he was under the impression that it had been decided that the whole bone develops from the proximal end of the hyoid arch—that it is one structure; the footplate does not develop separately from the rest of the stapes.

DR. HASKIN said that an especially interesting point about this case was that the boy had no zygoma. The lack of development of the lower jaw probably resulted from the fact that, as there was no glenoid fossa, the child had never been able to use that side of the jaw for mastication, and there had been no stimulation to produce the growth. He further said that he had looked up many histories of microtia cases, and had not found any described as in any way resembling this one. The position of the cartilage, well down on the cheek, rather than in the normal position, is the chief peculiarity. He had made pressure over where the internal ear should be, to see if

there was any vertigo, but had not succeeded in getting any response. There were a great many interesting points brought out by a study of the plates.

Paper: Clinical Observations of a New Iodin Preparation (Iodosan)

By M. D. Lederman, M. D.

DISCUSSION.

Dr. Heller said that all had at some time felt the need of having a good powder to blow into the ear for the treatment of chronic discharge, but had been limited to boric acid, which was nontoxic but had the unfortunate quality of not dissolving very rapidly, and consequently caking in the canal. The other antiseptics, bichlorid of mercury, iodin, and nitrate of silver, do not lend themselves to being used as powder; argyrol has been used frequently, but it is a nasty preparation, has little therapeutic value, and makes everything look black and as disgusting as possible. He had used this iodin powder, described by Dr. Lederman, on five or six cases, and felt that it was a distinct help in the treatment. It is readily absorbed. In one instance in which he used it in full strength there was a fair sized perforation, and the patient had a little pain, and nothing was done to counteract it; the man returned in an hour and said that he could not stand the pain, and on looking at the ear there was none of the powder left—it had all been absorbed excepting a little on the skin and outside of the meatus. Dr. Heller said that he was absolutely sure that it was absorbed, and was also sure that it was iodin, for he had had a little of the powder at home, and failing to realize that it ought to be kept in a glass stoppered bottle, found that the cork had been disintegrated at the lower end and had fallen out. He then showed a bottle where the cork had been used for only a few hours and that it was distinctly injured and discolored by the iodin. He repeated his belief that the powder was a valuable addition to the therapeutics of the ear.

Dr. Otto Glogau said that he had been using this preparation in chronic suppurations of the middle ear and maxillary antrum for over two years. Some of the patients complained of pain when a ten per cent mixture was used, so he employed

instead a three per cent mixture, and had had no trouble since It was necessary in using it to remove first all diseased tissue such as granulations, polypi, necrosed ossicles, etc. In chronic suppuration of the maxillary antrum he had used it after operation and found it very valuable. It becomes absorbed very rapidly and does not cake up the wound. Combined with surgical treatment in these chronic middle ear suppurations, this powder will be a very valuable aid in our therapeutics.

DR. CARTER asked whether the preparation was a mechanical mixture or a chemical compound. He also asked Dr. Lederman how he accounted for the fact that while there is only five per cent of iodin in the preparation, the remaining boracic acid powder does not cake as when plain boracic acid is used.

DR. LEDERMAN said that he was not enough of a chemist to describe the exact technic in the manufacture of the preparation, but that it was probably more of a mechanical than a chemical combination, as the iodin is very loosely bound with the boric acid and separates from it very readily, which is of especial advantage. In other preparations the iodin is so tightly bound that it is not readily liberated. This condition must be borne in mind in applying the powder, and one should be sure that the powder is finely divided before being blown into any cavity.

In a case still under treatment, the patient was a young man who had had a chronic suppuration for a number of years. In one ear, two-thirds of the drum was gone with granulation tissue present. The powder seemed to have some action on the granulation tissue in the middle ear, and when a cotton covered applicator was passed over the granulation tissue it was found to be friable and was easily removed. He has been under treatment for six weeks, the discharge in one ear has ceased and the drum has healed over. In this ear the perforation was at the posterior margin and involved the posterior third of the membrana. The other ear is almost dry.

The advantage claimed for the powder is that the iodin content is easily given off, and there is no danger of retarding the drainage. He has used it in a number of mastoid wounds, and found that it hastened the healing. It had also been used by a surgeon in vaginal gonorrhea, and in two instances it

had been used on a suppurating breast, and in a few applications the fistula which the surgeon had been trying to combat were healed. It has been used abroad in cases of suppurating antrums with very good results. The preparation is not yet on the market, but can be obtained in small quantities at Nauheim pharmacy. Five per cent has been found to be sufficiently strong to be effective, and in fact he generally uses a two per cent mixture, which seems to do the work very satisfactorily.

DR. LAW asked if an X-ray plate had been made after the condition cleared up, in the case mentioned.

DR. LEDERMAN said that the patient lived out of town and had not been seen for some time, and no plate was made after the discharge ceased.

DR. HASKIN suggested the employment of a very strong salt solution in cases of chronic suppurative ears, letting it remain in the ear. He had done this in several cases, following the suggestion of Sir Almroth Wright in treating suppurative wounds, and the result was very remarkable. By filling the ear up with a strong solution of salt and letting it remain in for half an hour, old cases of foul smelling ears would clear up very surprisingly. If the solution disappears through the eustachian tube, fill it up again.

Paper: Report of Thirty-seven Cases of Perisinus and Extradural Abscess—Lantern Demonstration.

BY H. B. BLACKWELL, M. D.

DISCUSSION.

DR. HAYS said that the speaker had not presented anything especially new on the subject of perisinus or extradural abscess, but only more about an operation which he has been performing for a good many years. No one questions the fact that a majority of the perisinus and extradural abscess cases are what might be termed latent, having existed for a considerable length of time, varying from four to six weeks or more —all of those in which there is a diminution of the acute symptoms and the diagnosis is made on the general looks of the patient, often corroborated by the X-ray picture. All agree that the most serious cases are those that have extended over

a considerable length of time and show destruction of the bone, the sinuses or the dura. A majority of cases get well, and Dr. Hays said that in discussing the subject at a former time, he had said it would seem that although the condition had continued until there was considerable destruction of bone, the bacteria apparently became attenuated in virulence. Often these cases heal more quickly than the acute cases, which do not heal up in two or three weeks.

Dr. Hays said that Dr. Blackwell was exceedingly fortunate in having such excellent results, losing only one patient out of thirty-five cases. In regard to the question of operation, Dr. Hays said that six or seven years ago he had described an operation which he had termed primary suture of the mastoid wound. At the time he was very severely condemned for doing it. It consisted mainly of closing the wound and putting in a wick of gauze, but the results have justified the procedure in certain cases, and the cosmetic results have been excellent. Anyone who has Dr. Blackwell's dexterity can go down into the attic and expose the malleus and the incus, but that is unnecessary in the majority of cases—and the best evidence of that is found in the cases he had reported, in which he had never seen the head of the malleus. In acute cases of mastoiditis, we should let the aditus and the attic alone as much as possible. The main destruction of bone is in the mastoid cavity, and if that is cleaned out thoroughly and drained by a large wick of gauze, the case will get well nicely.

Dr. Page complimented Dr. Blackwell on his excellent results, and said in regard to closing the wounds, however, he had had an experience similar to that reported by Dr. Hays. This occurred with a simple mastoid operation performed for mastoiditis with a perisinus abscess in a young man. His wound was closed down to a small drain at the lower end. The drain was removed in three or four days, and there was union and an apparently clean cavity at the end of a week. He was allowed to get out of bed on the fifth day and allowed to leave the hospital on the seventh day, not having had any rise in temperature. He was directed to report to the hospital in two days, but to return the next day if he felt ill. The next day he felt ill, but stayed at home, and on the day following came to the hospital with a temperature of 102°. He was

readmitted to the hospital for exploration of the wound, which, to external appearance, was free from inflammation. The skin flaps which had been reflected and replaced at the time of operation were found adherent to the sinus wall, and localized areas of infection were walled off by adhesions, with no communication with the lower angle of the wound. An infected clot was in the sinus, and it was found necessary to resect his jugular. Recovery occurred only after a stormy career with a femoral phlebitis and other complications. Dr. Page said this had cured him of taking any chances simply to get a rapid recovery.

While he closes the wound down to the lower angle, he protects the whole exposure of the dura and sinus to the farthest angle of the wound with gauze strips saturated in salt solution, with their ends at the lower angle of the wound, in order that no areas of infection can become walled off by adhesions between the integument and the knee of the sinus. More recently he has been using Dakin's solution in the place of normal saline, and the gauze is kept in place not longer than three days, and it may be removed the day after the operation if conditions appear unfavorable. For the next five or six days the wound is dressed and the extremities of its cavity are examined through a large speculum, and if everything is clean and there is no discharge from the canal, the skin is allowed to grow together at the end of a week or two. At the first indication of infection in the wound, however, it is opened sufficiently to properly avoid danger to the patient.

Dr. Page said, however, that he had never had such a percentage of success as that reported by Dr. Blackwell, in which all thirty-five cases (if he understood him correctly) healed within from fourteen days to three weeks. This was to him a very remarkable record.

Dr. Lederman said that some seventeen or eighteen years ago he had heard Dr. McEwen discuss the treatment of brain cases at the otological meeting in London, and had been deeply impressed by his remarks on the handling of the dura. McEwen stated that he did not remove the granulations on the dura, as they were a protection against further infection. Ever since then, in operating a perisinus or extradural abscess, he has carefully avoided injuring the barrier which nature has set

up, which is frequently destroyed in the attempt to make a clean wound. In operating upon acute mastoiditis cases, he is still satisfied to clean out the antrum and adjacent cells which are involved, but does not attempt to disturb the aditus or middle ear. If the antrum and mastoid cells are thoroughly cleaned, the middle ear condition will promptly subside.

DR. HERZIG complimented Dr. Blackwell on the excellent results secured, and asked how much time was allowed to elapse after doing a radical case before the first dressing was made—i. e., closed the wound. He also wished to know if he was correct in understanding Dr. Blackwell to say that the wounds healed in fourteen to twenty-one days, and how often the dressing was replaced.

Dr. Herzig said that he had sewed up three cases, closing the wound completely, with the exception of a small drain. One of these healed in four weeks. That one was found years ago. In the second case, a double mastoid, one side did not heal at all, the other side healed in four weeks. The third case did not heal for three months, but finally closed up. He wished to know how often Dr. Blackwell replaces the dressing, and if he packs it up to the antrum. The closed wounds in all of these cases show a strong covering, and no sinking in to any extent.

DR. HASKIN said that Dr. Blackwell was certainly to be congratulated upon his excellent results. The paper was really devoted to two questions—the perisinus abscess and the method of operation. Dr. Haskin said he was inclined to doubt the wisdom of opening the attic in acute cases. After opening and securing drainage, it would seem much safer, in the great majority of cases, to keep out of the attic, for the attachments of the incus to the aditus are very easily injured, and there does not seem any advantage in stirring up avenues of infection. In the great majority of cases, when the infection is removed from the mastoid process the condition will subside.

Dr. Haskin said that he employs another method of healing wounds, which to some might seem rather radical, but that he never sews up wounds in either actue or radical mastoid operations. He makes as small an incision as possible, puts in only what dressing is absolutely necessary, and keeps the

wound open for five or six days, and then cleanses the wound every day for a few days until the bone is covered with granulation tissue, and then leaves off all dressings. It is astonishing how rapidly they heal under this treatment, and how beautifully the edges heal without any depression or deformity. He has never had any bad results from this method of treatment. In the radical mastoid operation he employs the same procedure, taking down the canal wall clear to the ridge, splitting the narrow end of the canal about one-half inch, and packing the ends wide open, leaving the wound open behind, just as in a mastoid wound. After the bony wound has become thoroughly granulated, all the dressing is left off, and you are perfectly sure that the flap ends at the inner ends of the canal are adhered. When you look into the ear afterward, it is normal, so far as the canal is concerned. Dr. Haskin said it was his experience that patients so operated upon have better hearing and heal more rapidly. In the great majority of cases they are dry in three to four weeks.

Dr. Hays said that when he had presented the question of primary suture of mastoid wounds some years ago, he had made a remark that holds good today. In these cases of simple mastoid, where there is no exposure of dura or sinus, he closes up the entire wound except the lower angle. Only one small piece of packing is inserted to reach from the antrum along the posterior wall to the lower edge of the incision. This packing is removed at the end of thirty-six hours. No packing is used after this, but each day a probe is passed into the wound up as far as the antrum. The secretions in the mastoid cavity are expressed by massaging the wound from above downward.

Dr. Hays said that if any otologist can convince him that he is any better off for looking at the wound every day, and interfering with what nature is trying to do, he would stop this procedure. It is a very simple matter to open the wound, even on the fifth or sixth day, so that we can inspect the parts underneath. The trouble is we do not allow nature to do enough for us.

Dr. Hays said that many of his cases in which he had performed the simple mastoid operation had completely healed up on the ninth or tenth day, so that a small strip of adhesive plaster was all that was necessary over the wound.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of March 9, 1917.

Demonstration of Anatomic Specimens of the Temporal Bone.

Dr. Wm. M. Dunning expressed his gratification at the opportunity afforded him of exhibiting "his bones" to the members of the section, though he hardly felt that he could add anything of interest to their knowledge of the anatomy of the temporal bone. His part of the program was merely an exhibit of prepared specimens of the temporal bone, but the collection was somewhat unique in that it covered all the stages of growth from infancy to adult life, showing the appearance of the facial nerve on the surface at different ages. At birth the facial nerve emerges posterior to the drumhead and extends downward in a shallow groove as the mastoid develops, and usually occupies its normal adult position at the age of six.

The collection consisted of about sixty bones, which had been assembled during a period of some fifteen years. Dr. Dunning directed especial attention to the dissections of the semicircular canals and the various and unusual developments of the mastoid cells, some of them extending far forward and others into the occipital condyle. As a rule, at birth there are no mastoid cells except the antrum, which is sometimes as large as in the adult. The cells are probably developed between the first and sixth years, although Gray claims that they continue to develop until puberty. It is also unusual to find small cells at the mastoid tip, as stated by Gray. Large cells are much more usual, and not "cancellous bone containing marrow."

The collection was on exhibit, and the bones would speak for themselves. The first group—including sixteen bones—showed the development of the temporal bone from birth to adult life, and the position and size of the facial nerve at advancing ages. The next group represented different operations, and included some twenty specimens. The remaining specimens illustrated different anatomic conditions of the mastoid, and included a number of interesting instances of fracture.

Presentation of Specimens of Temporal Bone, Excavated to Show the Relations of Important Structures Encountered in Performing the Mastoid and Labyrinth Operations.

Presented by Dr. W. A. Scruton for Dr. A. B. Duel. The main points which Dr. Duel wished to bring out in presenting these specimens were the following:

The relation of the styloid foramen to the mastoid tip in infancy, childhood, and adult life. The series of bones, mounted in sequence from the newborn to adolescence, show with great emphasis the real danger of injury to the facial nerve which confronts the operator in elevating the periosteum in the region of the tip in young children.

A specimen is shown of the temporal bone in the newborn. In this it may be noted that the petrous and squamous portions are separate, also the ring. By reason of this multiple genesis, dehiscences are known to exist even in bones which have reached the period of total ossification; and under such circumstances it is quite conceivable that the simple operation of opening the membrana tympani (myringotomy) may deal a death thrust in the dura or the jugular bulb. Dr. John R. Page has reported a case of injury to the jugular bulb in myringotomy before this section.

The specimen consisting of the entire half of the head with the contents of the temporal bone worked out in detail is recommended for teaching purposes. The student may place this upon a table and by placing himself in position for operating, a study may be made of the planes and angles of approach to the landmarks in the position in which they will be found in actual practice. Study of small specimens only, viewed from all aspects, is likely to result in confusion of the student's orientation when he looks for the landmarks in the sequence of operative procedure.

The specimen consisting of the entire head containing a wax model of the brain is invaluable for teaching purposes, because the temporal bones, which are worked out in detail, are removable, permitting a view of the areas of the brain corresponding to any portion of the mastoid field.

Other specimens presented show the internal ear cut out in various fashions to show some particular detail.

DISCUSSION.

DR. HASKIN said that one of the points brought out in the specimens presented was the position of the facial nerve in regard to the tip. It comes out almost on a level with the superior wall of the canal during infancy, and it is very common for the facial nerve to be injured at that point in operations upon children. In studying mastoiditis he had always been especially impressed with two or three points, one of them being the fact of the depth of the cells between the sinus and the posterior canal wall—the depth to which they extend down into the mastoid. In some large mastoids these cells continue on, under or above the jugular bulb, and may extend clear through into the condyloid process or the occiptal bone. In his collection were cells where a pencil could be passed through to a condyle. Most operators in doing a mastoid take off the tip cortex, but do not take off much of the floor of the tip, yet examination of the bones shows that the cells extend very deeply beyond that point, and in severe cases these parts are not cleared out deep enough, for fear of injuring the facial nerve.

Another interesting point is the relationship of the jugular bulb with the middle ear and with the labyrinth. In the majority of cases one finds the jugular bulb is in contact with the floor of the labyrinth itself. There is very little if any space between the labyrinth and the wall of the jugular bulb. It is remarkable to realize the great number of cases in which this is so.

Another important point is the position of the facial nerve. Dr. Berens possesses the only specimen that Dr. Haskin has ever seen that has the facial nerve going obliquely across the tip of the mastoid; in operating upon this case the nerve was severed. Dr. Duel has several specimens showing dehiscences in the facial canal of the middle ear. That is not uncommon. Quite a large proportion of cases have dehiscences in the facial canal just at the ridge. To a man who has not done much surgery in the mastoid, there is more danger in the descending portion of the facial, for the canal is exposed, and if one gets too close to the canal wall in following these numerous and deep cells, he is liable to injure the nerve in the

descending portion; everyone looks out for the aditus and avoids it.

In children the facial nerve at first comes out near the superior wall of the annulus, and one watches for the development of the mastoid tip, the facial canal becomes deeper, or rather the mastoid develops over it and protects it.

Another point is the close contact of the jugular bulb with the floor of the tympanum in many instances. The roof of the bulb is really the floor of the middle ear.

Still another important point is the question of the carotid canal, which forms the floor and part of the anterior wall in the tympanum and the middle ear. By holding the specimens up to the light, one can see the dehiscences in the floor of the carotid-canal, which show how easily one could puncture the artery in these positions.